ASSESSMENT OF LEFT ATRIAL FUNCTION BY TISSUE DOPPLER STRAIN IMAGING IN MITRAL STENOSIS BEFORE AND AFTER BALLOON MITRAL VALVOTOMY

A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR DM (BRANCH II, CARDIOLOGY) EXAMINATION OF THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI TO BE HELD IN JULY / AUGUST 2010

BONAFIDE CERTIFICATE

This is to certify that the work presented in this dissertation titled "**Assessment of Left Atrial Function by Tissue Doppler Strain Imaging in Mitral Stenosis Before and After Balloon Mitral Valvotomy"** done towards fulfillment of the requirements of the Tamilnadu Dr. MGR Medical University, Chennai, for the DM- (Branch II) (Cardiology) examination to be conducted in July/August 2010, is a bonafide work of the candidate Dr. Alok Sehgal, Senior Post-graduate student in the department of Cardiology, Christian Medical College, Vellore under my guidance & supervision. This dissertation has not been submitted, fully or in part to any other board or university.

Dr. V Jacob Jose
M.D., D.M., FACC, MS, FCCP, FIAE
Professor, Department of Cardiology,
Christian Medical College,
Vellore - 632004

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Dr. Sunil Thomas Chandy
M.D., D.M., FCSI, FIC(Aus)
Professor and Head,
Department of Cardiology,
Christian Medical College,
Vellore - 632004

DECLARATION

I, Dr Alok Sehgal hereby declare that this dissertation entitled '**Assessment of Left Atrial Function by Tissue Doppler Strain Imaging in Mitral Stenosis Before and After Balloon Mitral Valvotomy**' has been prepared by me under the direct supervision and guidance of Dr. V Jacob Jose, Professor, Department of Cardiology, Christian Medical College, Vellore. This is being submitted to Dr M.G.R medical university in partial fulfillment of regulations for the DM (Cardiology) examination to be held in 2010.

This dissertation has not been submitted by me either in part or in full on any previous occasion to any university or institution for the award of any degree or diploma.

Place: Vellore

Dr. Alok Sehgal
Postgraduate student
Department of Cardiology
Christian Medical College
Vellore

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CONTENTS

ASSESSMENT OF LEFT ATRIAL FUNCTION BY TISSUE DOPPLER STRAIN IMAGING IN MITRAL STENOSIS BEFORE AND AFTER BALLOON MITRAL VALVOTOMY

ABSTRACT

Assessment of Left Atrial Function by Tissue Doppler Strain Imaging in Mitral Stenosis Before And After Balloon Mitral Valvotomy

BACKGROUND: Assessment of regional Left Atrial function may provide insight in atrial electromechanical remodeling. New echocardiographic techniques, such as Tissue Doppler imaging and Strain (rate) imaging allow a non invasive measurement of regional function of the myocardium. Both techniques have been well validated for assessment of regional LV function. Recently these techniques have also been used for the assessment of Left Atrial function.

AIMS: To assess Tissue Doppler Velocities, Strain, Strain rate variation of Left Atrial lateral wall and Interatrial septum in patients with severe Mitral Stenosis and effect of Balloon Mitral Valvotomy on measured parameters.

METHODS: Twenty five symptomatic patients with severe Mitral Stenosis were evaluated by Echo, Doppler studies along with Left Atrial function by Tissue Doppler Imaging for velocity, Strain and Strain Rate measured at mid point of Interatrial septum and Left Atrial Lateral wall. All patients were assessed before and 24 hours after Balloon Mitral Valvotomy. 25 age matched controls were also evaluated.

RESULTS: Pulsed Tissue Doppler velocities, E' and A' of Inter Atrial Septum were lower in patients of severe Mitral Stenosis (E'=6.9±3.5 vs 9.8±2.7cm/sec,p=0.002;A'=7.1±2.6 vs 9.2±2.9 cm/sec,p=0.01).Left Atrial Lateral wall velocities were also statistically less in Mitral Stenosis as compared to controls (E'=6.6±1.9 vs 15.9±4.3cm/sec,p<0.001; A'=7.4±2.9 vs 13.1±3.6 cm/sec,p<0.001). Lateral E' velocity improved significantly after Balloon Mitral Valvotomy (P<0.001). Left atrial Strain at ventricular end systole was lower in patients of Mitral Stenosis when compared to controls as measured at Inter Atrial Septum (11.4% ± 6.3 vs 29.6%±10.5, p<0.001) and Left Atrial lateral wall (18.2%±8.8 vs 28.4%±14.0,p=0.004). IAS systolic strain improves significantly after Balloon Mitral Valvotomy (18.0%±10.6 vs 11.4% ± 6.3,p=0.02) and that measured at Left Atrial lateral wall showed a trend towards improvement (22.8%±10.6 vs 18.2%±8.8,p=0.07).

CONCLUSION: Left Atrial function as assessed by Tissue Doppler velocities and Tissue Doppler derived Strain is lower in patients with severe Mitral Stenosis. Left atrial reservoir function assessed by Strain imaging improves within 24 hours after Balloon Mitral Volvotomy.

INTRODUCTION

Normal Left Atrial function consists of reservoir, conduit and pump function [1,2]. Reservoir function occurs during left ventricular systole when the Mitral valve is closed, Left Atrium is relaxed, and the Mitral annulus is temporarily displaced toward the apex [3]. Left Atrium acts as a conduit in diastole, when the Mitral leaflets open and allow blood to enter the left ventricle. At the end-diastole, the left atrium contracts, and the pump function occurs [4]

In Mitral Stenosis Left Atrial function may be disrupted because of increased Left Atrial afterload. Rheumatic Mitral Stenosis is associated with considerable fusion of the commissures and reduction of mitral valve area and leaflet mobility [5,6]. Moreover there is marked increase in Left Atrial dimension and consequently, an impairment of Left Atrial pump function [5]. Finally when atrial fibrillation ensures, complete loss of Left Atrial pump function occurs as a result of cessation of atrial systole [7].

Many methods have been used previously to assess atrial function, both invasive and non invasive. However they are either difficult to be applied, time consuming, inaccurate as they are load dependent and difficult to reproduce because they are observer dependent. Moreover some of there indices do not accurately evaluate atrial reservoir function. [8,9]

Development of Tissue Doppler imaging has enabled to accurately evaluate myocardial properties in a load independent and reproducible manner. Tissue Doppler derived Strain Rate and Strain imaging has the advantage of being not affected by translational movements thus reflects actually the myocardial deformation.[10] Its Initial use has been for quantifying regional myocardial deformation in ventricles.[11, 12]

It has also been used to evaluate Left Atrial function in conditions like Hypertension[13], Diabetes[14], and post Cardiac Resynchronization Therapy.[15] Assessment of left atrial function in severe Mitral Stenosis using Strain and Strain Rate imaging has not been done earlier.

In this study Left Atrial function in patients with severe Mitral Stenosis was assessed by Tissue Doppler, Strain and Strain Rate imaging and compared with normal healthy individuals. Effect of Balloon Mitral Valvotomy on atrial function was also determined.

AIMS AND OBJECTIVES

1. To assess Tissue Doppler Velocities ,Strain and Strain Rate variation of left atrial Lateral Wall and Interatrial Septum in severe Mitral Stenosis

2. To evaluate the effect of Balloon Mitral Valvotomy on left atrial Tissue Doppler velocities and Strain parameters.

REVIEW OF LITERATURE

Introduction:

Rheumatic Fever and Rheumatic Heart Disease is a significant cause of cardiovascular diseases in the world today. Despite a documented decrease in the incidence of acute Rheumatic Fever and similar documented disease in the prevalence of Rheumatic Heart Disease in developed countries, it remains a major public health issue in countries like India.

Rheumatic Fever and Rheumatic Heart Disease are non-suppurative complications of Group A Streptococcal pharyngitis due to delayed immune response. Although all cardiac valves may be involved by the Rheumatic process, the Mitral Valve is involved most prominently and in virtually all cases.

Epidemiology:

After Human Immunodeficiency Virus, Tuberculosis and Malaria, Group A Streptococcus has a global mortality range comparable to that of the pathogens causing Hepatitis, Measles, Haemophilus Influenza[16]. It was estimated in 2005 that approximately 15.6 million people had Rheumatic Fever or Rheumatic Heart Disease, with sub-Saharan Africa and South Central Asia accounting for majority of cases[16]. The prevalence of Acute Rheumatic Fever /Rheumatic Heart Disease in India has been reported to vary from very infrequent to very high depending upon the source of information e.g. registry data, school prevalence data and hospital admissions.

School survey data from our country has reported a prevalence of Rheumatic Heart Disease to be as high as 4.54 per 1000 by Lalchandani et al[17] to as low as 0.5 per 1000 by Mishra et al[18]. The largest school survey conducted till date was from our institute conducted from 2001-2002. A total of more than 2 lac children between the ages of 6-18 years were screened and a prevalence of 0.68 per 1000 was reported by Dr. Jose et al[19].

Pathogenesis:

Although the pathogenesis of Acute Rheumatic Fever and Rheumatic Heart Disease remains somewhat elusive, Acute Rheumatic Fever is clearly the result of an exaggerated immune response to Group A Streptococcal pharyngitis[20]. The clinical manifestation of the response and its severity in an individual is determined by host genetic susceptibility, the virulence of the infecting organism and a conducive environment.

Initial Streptococcal infection in a genetically predisposed host and susceptible environment leads to the activation of T & B lymphocytes by Streptococcal antigens and superantigens, which results in production of cytokines and antibodies directed against Streptococcal carbohydrate and myosin . It has been proposed that injury to the valvular endothelium by these anti carbohydrate antibodies leads to an upregulation of VCAM 1 and other adhesion molecules [21]. VCAM 1 expression is a hallmark of inflammation and it heralds cellular infiltration. VCAM 1 interacts with very late antigen on activated lymphocytes and lead to the extravasation of activated CD4 and CD8

lymphocytes into the valve tissue. A break in the endothelial continuity of a heart valve by anti streptococcal antibodies expose subendothelial structures and lead to a chain reaction of valvular destruction. Once the leaflet becomes inflamed, revascularization occurs and lymphocytes can infiltrate the valve both through the valvular surface endothelium from without and through the revascularization from within. This perpetuate the cycle of valvular damage leading to valvular scarring

Pathologic Evolution of Mitral Stenosis:

During acute rheumatic fever with carditis, involvement of the Mitral Valve consists of tiny, translucent nodules located along the line of closure of the valve, occasionally also involving subvalvular parts of the chordae. Microscopic sections of these nodules show largely nonspecific proliferation of fibroblasts and macrophages. These translucent vegetations later become opaque and grey, and eventually more of the valve leaflet becomes thickened. Changes within the valve structure involve deposition of fibrin upon the cusps with loss of the normal morphology, hyalinization, and eventually the covering of the leaflets with endothelium. This process may lead to fusion of the valve commissures. Brock [22] postulated that the initial point of fusion of the two leaflets occurred at the "critical area of tendon insertion," i.e., the point where the shortest and most direct chordae connect with the cusps. When fusion occurs at these points, portions of the cusps lateral to them are immobilized, thereby facilitating more commissural fusion. When Mitral Stenosis is fully developed, three distinct types have been recognized:[23]

1. *Commissural type*: consisting of fusion of the commissures with little involvement of cusps or chordae
2. *Cuspal type*: in which the leaflets are converted into stiff, rigid, leathery (later calcified) structures; and
3. *Chordal type*: in which the chordae are fused, thickened, and shortened, thereby interfering with the mobility of the leaflets.

In addition to the pure forms, combination of these types occurs. Various anatomic forms of mitral stenosis may affect atrioventricular filling in similar manner.

Pathophysiology of Mitral Stenosis:

The normal Mitral Valve orifice is 4 to 6 sq. cm, which essentially creates a common chamber between left atrium and left ventricle in diastole. In very early diastole, there is a brief, small gradient between left atrium and left ventricle, which rapidly dissipates so that pressure in the 2 chambers is equal for most of the filling. As the Mitral orifice narrows in Mitral Stenosis, it curtails free flow of blood from left atrium to left ventricle, and a pressure gradient develops between the 2 chambers.

This pressure gradient is added on to left ventricular diastolic pressure, which results in increasing left atrial pressure that eventually leads to left atrial enlargement and pulmonary congestion. As stenosis severity worsens, flow restriction limits left ventricular output. Pulmonary congestion and reduced cardiac output mimic left ventricular failure.

Since it is primarily the left atrium that generates the force necessary to drive blood across the stenotic Mitral Valve, Mitral Stenosis causes left atrial structural and functional abnormalities. These left atrial abnormalities like left atrial enlargement may lead to various complications associated with Mitral Stenosis like atrial fibrillation, or predisposition to left atrial clot formation. Thus it is essential to understand the importance of Left Atrial function in Mitral Stenosis and methods to assess it.

ASSESSMENT OF LEFT ATRIAL SIZE, ANATOMY AND FUNCTION

Left atrium modulates left ventricular filling through three components, a phase of reservoir or expansion during systole, a conduit phase during diastole and an active contractile component during late diastole. This active contractile component of left atrium has an important role in patients with ventricular dysfunction as a 'booster pump' to augment ventricular volume. Augmented left atrial booster function is one of the mechanisms compensating for decreased early filling in patients with reduced left ventricular compliance, whereas a loss of atrial contraction, as a result of atrial fibrillation or ventricular pacing, reduces cardiac output by approximately 15 -20% [24,25].

During exercise left atrial reservoir and booster functions are augmented, whereas conduit function is not increased reservoir function may play an important role in accelerating left ventricular filling by helping to maintain an enhanced atrioventricular pressure gradient during diastole and also by

increasing left atrial booster function through an increase in preload[26]. An isolated decrease in left atrial compliance is associated with relative increase in the conduit function of the left atrium. The ability to optimally redistribute left ventricular filling among reservoir, conduit and booster pump functions in a potentially important adaptation that may occur in left atrium in response to changing hemodynamics[27].

Causes and Mechanism Of LA Dilatation

In large population based studies, it has been demonstrated that Left Atrial size is an important predictor of cardiovascular outcome[28-30]. Tsang et al[30] recently demonstrated that a larger indexed Left Atrial volume predicted a higher risk of cardiovascular events after adjustment for age, gender and other covariates. Patients with a severely increased left atrium are at the highest risk for the development of cardiovascular events.

Left atrial dilatation can occur in a broad spectrum of cardiovascular diseases including hypertension, left ventricular dysfunction, mitral valve disease and Atrial Fibrillation. In general two major conditions are associated with left atrial dilatation; pressure overload and volume overload[31]. Left Atrial volume overload frequently occurs in the setting of mitral regurgitation. Pressure overload is most frequently caused by an increased LA after load, secondary to mitral valve disease or left ventricular dysfunction. Atrial fibrillation is another important factor associated with Left Atrial dilatation. Atrial fibrillation is most commonly encountered cardiac arrhythmia, and the association of Left Atrial

enlargement and Atrial Fibrillation has been well recognized[28, 32-35]. However whether Atrial Fibrillation causes Left Atrial dilatation or vice versa still remains controversial. Severe studies suggest that Left Atrial enlargement may cause Atrial Fibrillation [28,32,33]. In Framingham Heart study[32], M-mode derived Left Atrial size was an independent risk factor for development of Atrial Fibrillation. Tsang et al[28] demonstrated that Left Atrial volume was a strong predictor of Atrial Fibrillation, incremental to clinical risk factor. However other studies have revealed that Left Atrial enlargement may be the consequence of Atrial Fibrillation [34, 35].

Importance Of Left Atrial Size/Anatomy Assessment

Assessment of Left Atrial size is important since it has been shown to provide strong prognostic information. The incremental value of Left Atrial size over conventional risk factors has been demonstrated in several studies [30,36-38]. In Framingham Heart study[38] it was demonstrated that Left Atrial enlargement was significant predictor of death in both men and women.

In particular, assessment of Left Atrial size is important in patients with Atrial Fibrillation. The guidelines on management of patients with Atrial Fibrillation recommend a standard two-dimensional and Doppler echocardiogram with assessment of Left Atrial size and function, in the clinical evaluation of all patients with Atrial Fibrillation [39]. Orange et al [37] demonstrated the predictive value of Left Atrial dilatation in patients with lone Atrial Fibrillation. In this population based study with a median follow up of 27 years, it was noticed that in

patients with lone Atrial Fibrillation, Left Atrial volume was a strong predictor of adverse events independent of age and clinical risk factors [37].

MULTIMODILITY IMAGING OF LEFT ATRIUM

Echocardiography:

For assessment of Left Atrial size various echocardiography techniques are available, including transthoracic, transesophageal and intracardiac echocardiography. Transthoracic echocardiography is most commonly used in daily clinical practice to assess Left Atrial size.

Transthoracic echocardiography:

Feigenbaum[40] was the first to demonstrate the correlation between Left Atrial dimension assessed with M mode echocardiography and angiographic Left Atrial size. The Left Atrial anterioposterior diameter as assessed with M-mode is most commonly used in daily clinical practice and in large studies. However M mode is not sufficient to determine true Left Atrial size, since it represents only one dimension of the Left Atrium[26]. In particular in Left Atrial enlargement, which may result in an asymmetrical geometry of the Left Atrium, M-mode echocardiography may under estimate the Left Atrial size. Therefore optimal assessment of LA size should include Left Atrial volume measurements [41, 42]

Various methods for assessment of Left Atrial volume with 2D echo are available, including the cubical method, area- length method, ellipsoid method and modified Simpson's rule (figure 1). In an prospective study including 621 patients,[43] it was demonstrated that the biplane area-length method and Simpson's method compared closely, whereas ellipsoid method systematically under estimated Left Atrial volume

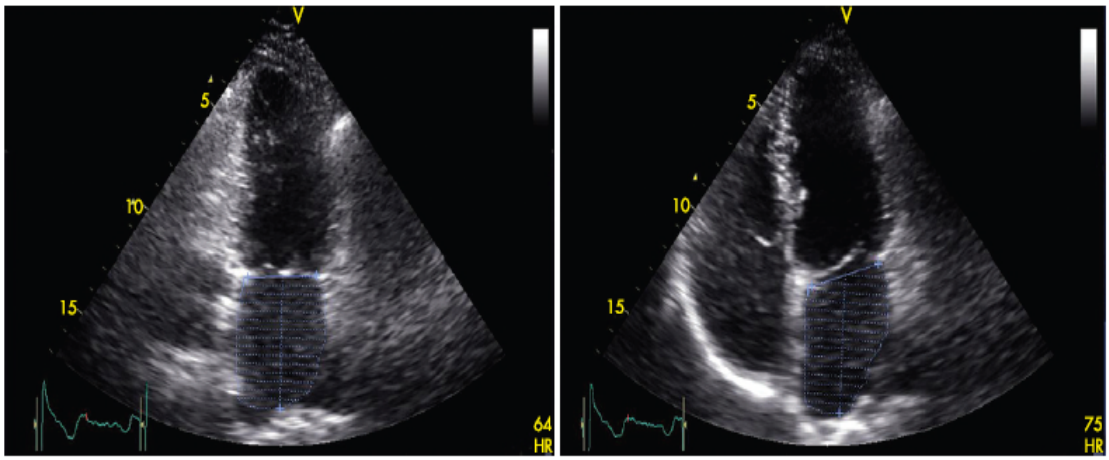

Figure 1: Measurement of Left Atrial volumes with transthoracic echocardiography using the modified biplane Simpson's rule. The maximum Left Atrial volume is assessed during ventricular systole in the apical two-chamber (left panel) and apical four-chamber (right panel) views. Maximal Left Atrial volume was 46 ml; minimal Left Atrial volume was 22 ml, resulting in a Left Atrial ejection fraction of 53%.
Ref: Reproduced from Heart 2007; 93:1462 [44]

. Recently 3-dimentional echocardiography has been introduced. A number of studies have demonstrated the feasibility of three dimensional echocardiography for assessment of Left Atrial volumes (figure 2)[45,46] and it has been validated against MRI[47]. Jenkins et al [45] demonstrated that three dimensional echocardiography allows accurate Left Atrial volume assessment with low test-retest variation, and a lower intra observer and inter observer

variability as compared to two dimensional echocardiography. However there still remain some technical limitations.

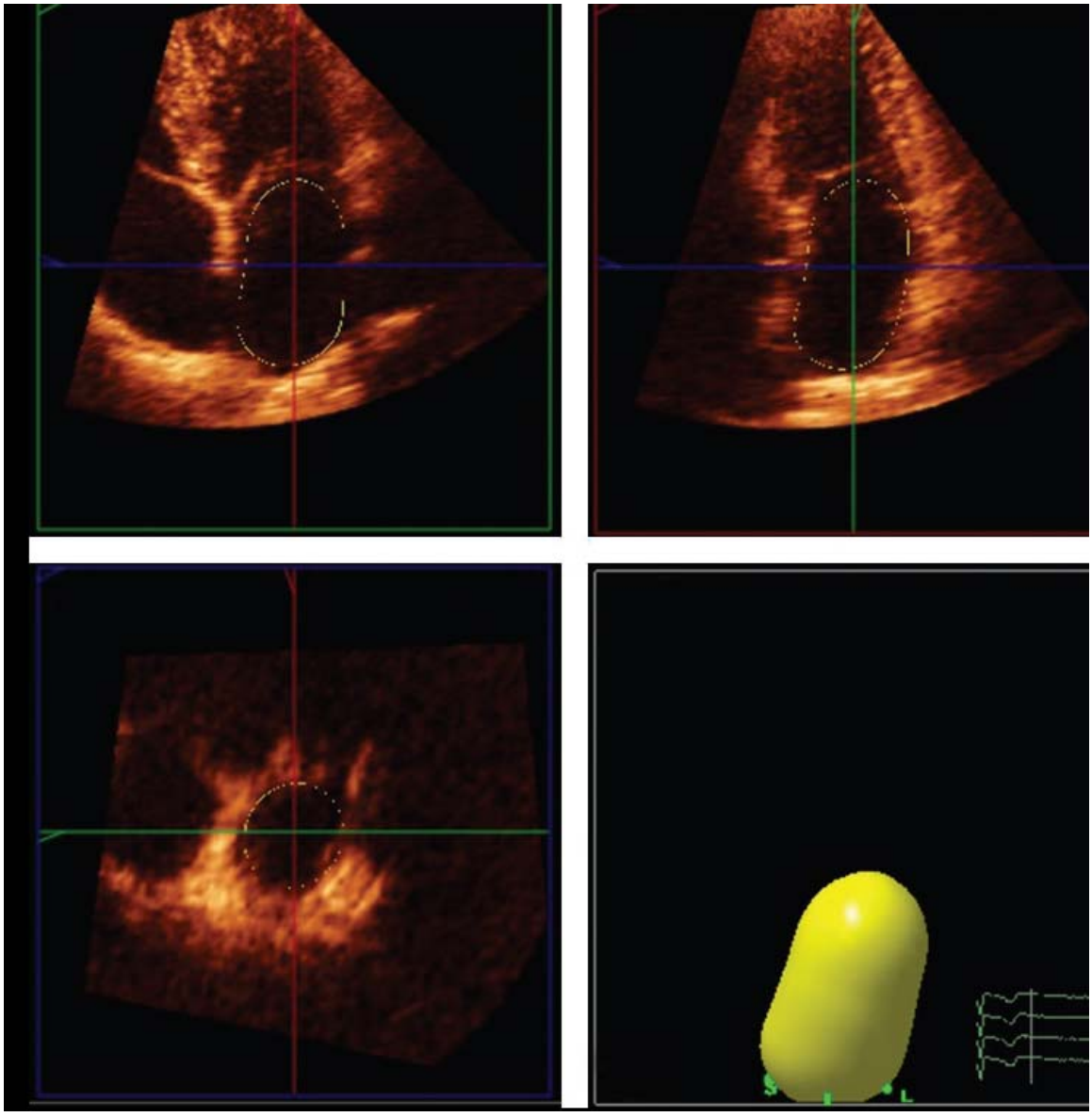

Figure 2: Real-time three-dimensional echocardiogram for the assessment of Left Atrial volumes. Panels A to C represent the coronal, sagittal and transverse planes, respectively. With the use of a five-point tracing algorithm, Left Atrial volumes can be obtained throughout the cardiac cycle, represented by the ''shell'' in panel D. In this example, Left Atrial maximum volume was 53 ml
Ref: Reproduced from Heart 2007; 93:1463 [44]

Transesophageal echocardiography:

Transoesophageal echocardiography provides good views on the Left Atrium and appendage. However, visualizing the complete left atrium to determine its size with transeophageal echocardiography may be hampered by the close proximity of the probe to the Left Atrium. As a result, measurements of Left Atrial size with transesophageal echocardiography have not been standardized.

Transoesophageal echocardiography is considered the procedure of choice for assessment of thrombi in the Left Atrial cavity or atrial appendage. In addition, transesophageal echocardiography is also helpful in assessment of Left Atrial appendage emptying velocities, which are correlated with thrombus formation (velocities <20cm/sec) with maintenance of sinus rhythm after cardioversion (velocity >40cm/sec) [48].

Intracardiac Echocardiography:

Intracardiac echocardiography is only used during interventional procedure, such as percutaneous closure of atrial septal defects and catheter ablation procedures. Therefore no standardized size and volume are available. During these interventional procedures, intracardiac echocardiography cans accurately visualized LA anatomy and related structures [49].

Furthermore Left Atrial function can be assessed with Intracardiac echocardiography. Rotter et al [50] demonstrated a good correlation between Intracardiac echocardiography and transeophageal echocardiography for

measurement of Mitral E wave velocity and Left Atrial appendage emptying velocity. Although Intracardiac echocardiography is limited by monoplane character and the lack of standardized measurements of Left Atrial size, it is a valuable tool for interventional procedures.

Multislice CT:

The application of multi-slice CT (MSCT) in cardiac imaging has rapidly expanded in the past few years. Since MSCT has an excellent spatial & temporal resolution, it can accurately quantify Left Atrial volumes by using the modified Simpson's method [51]. However because of the radiation exposure and the use of contrast agents, MSCT is not routinely used for the assessment of Left Atrial size.

Magnetic resonance imaging:

Magnetic resonance imaging (MRI) is considered the most accurate technique for the non-invasive assessment of atrial volumes, because of high spatial resolution and the excellent myocardial border detection. Detailed information of Left Atrial size and volumes throughout the cardiac cycle can be acquired with MRI. Similar to MSCT, a modified Simpson's method can be used to determine Left Atrial volume. However due to its relatively long acquisition times and the cumbersome data analysis, Left Atrial volume assessment with MRI is not performed in daily clinical practice.

ASSESSMENT OF REGIONAL LA FUNCTION:

Regional Left Atrial function is not routinely assessed, and therefore no standardized parameters are available. This can be partly explained by the fact that non-invasive evaluation of regional Left Atrial function may be hampered by the relatively thin Left Atrial walls. However, assessment of regional Left Atrial function may provide more insight in atrial electromechanical remodeling and may be helpful in management of Atrial Fibrillation with surgical or catheter ablation. New echocardiographic techniques, such as Tissue Doppler imaging and Strain (rate) imaging, allow non invasive measurement of regional function of the myocardium. Tissue Doppler imaging quantifies regional tissue velocities of the myocardium. Strain & Strain rate represent local tissue deformation and the rate (speed) of local deformation respectively [52]. Both techniques have been well validated for the assessment of regional left ventricular function. Several studies have applied these new techniques to assess Left Atrial function [53-57]

Tissue Doppler imaging allows quantification of regional myocardial velocities, and assessment of the timing of peak systolic and diastolic velocities of the myocardium (figure 3). Therefore it may quantify regional electromechanical Left Atrial function, such as total electromechanical activity of the atria (represented by the interval between the onset of P wave on the ECG to the end of the A wave on the tissue Doppler images)[54]. However, the clinical relevance and the exact correlation of these new Tissue Doppler derived

parameters of regional Left Atrial function with conventional parameters, such as Mitral inflow A velocity and Left Atrial volumes, needs further investigations. A limitation of Tissue Doppler imaging for evaluation of regional Left Atrial function is the angle depending of the technique.

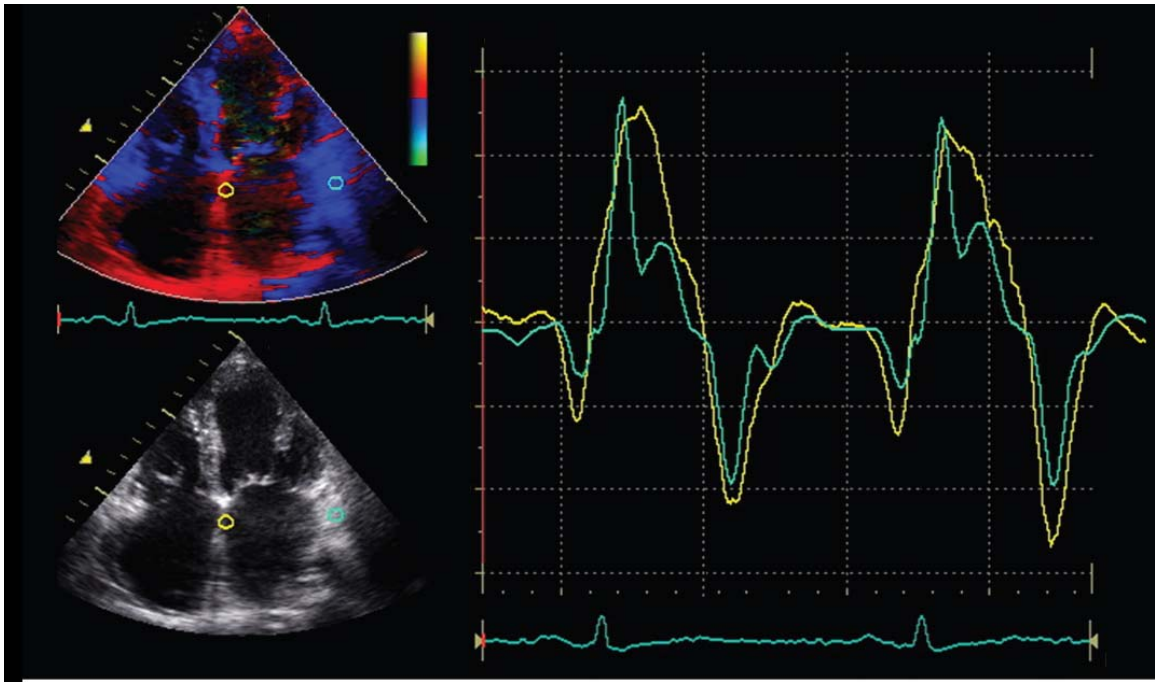

Figure 3: Color-coded tissue Doppler imaging in the apical four-chamber view for the assessment of regional Left Atrial function. The samples are placed in the basal atrial septum (yellow curve) and the basal atrial lateral wall (green curve). From the myocardial velocity curves, peak systolic and diastolic velocities can be assessed. Early diastolic filling is indicated by E' and late diastolic filling is indicated by A'.
Ref: Reproduced from Heart 2007; 93:1462 [44]

Strain and Strain Rate imaging are new tools for the assessment of regional myocardial deformation of Left Atrium [55]. In contrast to Tissue Doppler imaging, Strain imaging is not hampered by myocardial tethering. Furthermore Strain imaging allows for differentiation between active contraction and passive motion [52]. However thin atrial wall may not generate clear Strain curves and

therefore requires careful interpretation. Several studies have demonstrated the value of regional atrial Strain in the analysis of patients with Atrial Fibrillation undergoing cardioversion [56,57]. Di Salvo et al [56] studied 65 patients with Atrial Fibrillation and performed Tissue Doppler imaging of the standard apical images of the Left Atrium. It was noticed that all Tissue Doppler imaging derived parameters of Left Atrium, including Tissue Velocities, Strain and Strain Rate were significantly reduced in patients with Atrial Fibrillation, compared with healthy controls. The assessment of regional Left Atrial function by Tissue Doppler imaging or Strain imaging may be of value in the clinical follow up of patients with Atrial Fibrillation undergoing catheter ablation or cardioversion. However more studies are needed to appreciate the value of regional Left Atrial Strain and its role to guide use of medications in patients with Atrial Fibrillation.

Left atrial pressure – area relationship

In physiological investigations, the pressure area relation is the most accurate and representative index of left atrial hemodynamic status. Real time two- dimensional echocardiographic imaging with automated boundary detection to estimate left atrial area changes has been applied. To obtain left atrial pressure, a catheter tipped micromanometer is introduced retrogradely into the left atrium using streerable cardiac catheter.

The left atrial pressure- area relationship consists of two loops, the A loop representing left atrial pump function and the V loop representing left atrial reservoir function (figure 4). The areas of the A and V loops of the pressure area

relationship as well as the left atrial chamber stiffness constant can be calculated.

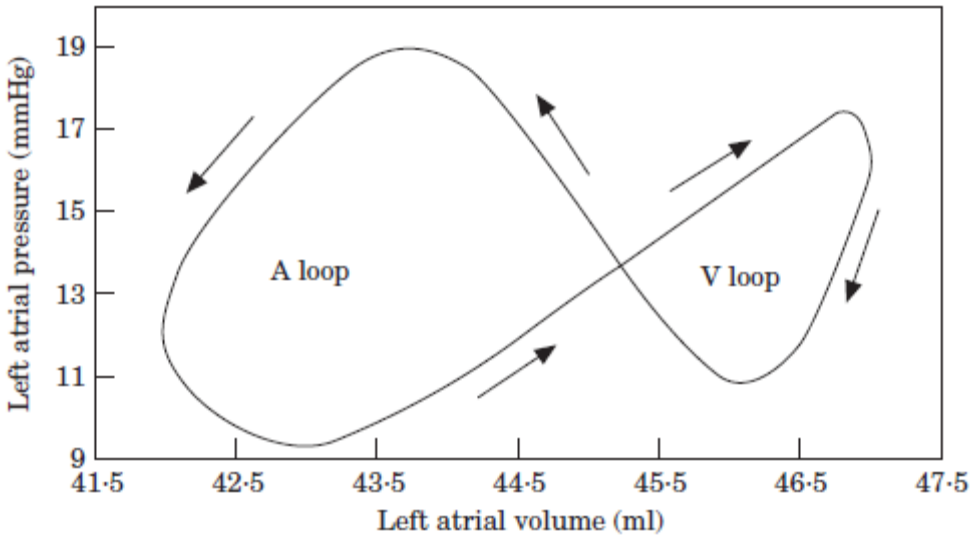

Figure 6: The left atrial pressure–volume relationship is composed of two loops: the A loop, expressing the left atrial pump function and the V loop, expressing the reservoir function of the left atrium. During the filling period, the curve is directed upward and to the right, and after maximal pressure and volume of the filling period have been reached, the curve turns clockwise and downward corresponding to the passive emptying, and subsequently, active emptying phases

Ref: Eur Heart J, Vol. 22, issue 1, January 2001; 26 [58]

The left atrial stroke works index can be accurately calculated, and a very good correlation has been found with left atrial preload. Left atrial stroke works index was found to be lower in patients with heart failure, whereas the left atrial stiffness constant was increased in patients with heart failure and atrial fibrillation compared with normal subjects. In addition, the increased inotropic state after dobutamine administration resulted in improved left atrial pump function (stroke works index) in normal subjects and patients with heart failure, as well as in decreased stiffness constants in all group of patients. This method is both safe and reproducible for obtaining the left atrial pressure-area relationship. Left atrial

function is impaired in patients with heart failure and in those with atrial fibrillation and may be acutely improved with inotropic agents in both normal and diseased atria[59].

Left atrial elastance:

Contractile function of the ex-vivo, isolated left atrium has been described by a time – varying elastance (E_t.), although this atrial chamber property has not been shown in vivo. In the intact heart, left atrial contraction may be approximated by time varying elastance with time dependant changes in E_t .Left atrial systolic pressure–volume relationship using either the non-isochronal maximum pressure to volume ratio or end systole may be useful as an estimate of Emax. They are highly linear and sensitive to calcium induced changes in inotropic state and may be useful in identifying left atrial chamber adaptation to chronic haemodynamic loads [60].

STRAIN AND STRAIN RATE IMAGING

Regional endocardial motion can be used to define regional myocardial function, is a long standing concept. This principle has formed the basis of many angiographic, nuclear and 2 dimensional (2D) echocardiographic studies [61-64]. A better description of regional myocardial function can be determined by local wall thickening and thinning characteristics, which are not necessarily directly related to endocardial motion. The most optimal data set which may describe regional

myocardial function would define wall deformation characteristics in three dimensions and in real time. Three dimensional (3D) local wall deformation can be acquired by Magnetic Resonance Imaging [65,66], while Computed Tomography and Gated Single Photon Emission Computed Tomography/ Positron Emission Tomography (SPECT/PET) imaging enable 3D acquisition of wall thickening & thinning [67,68]. However, none of these are real time techniques and the current temporal resolution used in clinical practice does not resolve all myocardial mechanical events. In contrast real time local thickening /thinning parameters can be obtained from ultrasound by grey –scale M mode recording, but only in one dimension and for limited number of regions of the myocardium [69]

Tissue Doppler myocardial imaging is an upcoming cardiac ultrasound technique which can resolve all means of myocardial velocities along its imaging lines. Initial clinical studies have examined the potential diagnostic role of this technique in determining regional myocardial function from velocity data sets for various diseases. Although encouraging data were obtained, it was clear that the interrogation of regional myocardial velocities alone has two major drawbacks. Firstly, since the amplitude of the estimated velocity is dependent on angle at which the region is imaged, accurate quantification of peak velocities can be difficult. Secondly, overall heart motion rotation and contraction of adjacent myocardial segments will influence regional velocity estimates[70]. In order to overcome some of these problems, ultrasonic Strain Rate imaging, or in other words, rate of deformation imaging, has been developed by estimating spatial

gradients in myocardial velocities. From Strain Rate curves, local strain (i.e. regional deformation curves) can be extracted, resulting in the concept of regional Strain imaging. Concept of Strain and Strain Rate imaging as measure of myocardial property was first introduced by Mirsky and Parmley et al [71]

STRAIN

Strain is defined as deformation of an object, normalized to its original shape. In an one dimensional object (i.e. an infinitesimally thin bar), the only possible deformation of the object is lengthening or shortening. This is illustrated in figure 5.

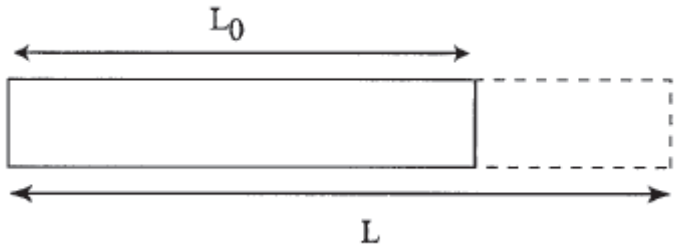

Figure 5: Deformation (strain) of a one-dimensional object is limited to lengthening or shortening. Strain is the deformation of an object relative to its original shape.
Ref: Eur J Echocardiography, 2000; 1:155 [74]

The relative amount of deformation is defined as Strain. Strain can thus be written as

$$\varepsilon = \frac{L - Lo}{Lo} \quad \ldots\ldots 1$$ [74]

with L the length of the object after deformation and Lo its original length. Since it is the change in length relative to its initial length, it is a dimensionless quantity

(often expressed in percent). By convention, lengthening is represented as a positive valve for strain, while shortening is represented by a negative value.

For a 2 dimensional object, the deformation is not limited to lengthening or shortening in one direction. It can lengthen or shorten along the X or Y axes (figure 6) and can distort by the relative displacement of the upper to the lower border or the right border to the left border.

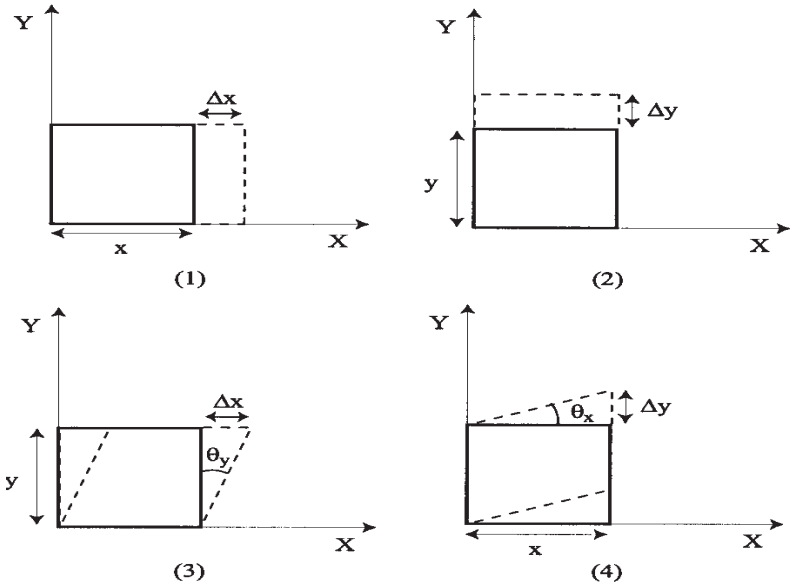

Figure 6: For a 2D object, deformation, i.e. strain, can be described by four strain components: two normal strains [(1) and (2)] and two shear strain components [(3) and (4)]. The shear strains are also completely characterized by the angles ɵx and ɵy.
Ref: Eur J Echocardiography, 2000; 1:155 [74]

The first type of deformation is called the normal strain (since its associated motion is normal to the border of the object), while the latter deformation is called the shear strain (since its associated motion is parallel to the border of the object). In order to describe the deformation of a 2 dimensional object completely, all four strain components have to be known.

The most general situation is that of a 3D object which deforms (as in a myocardial segment). In this case, there are 3 normal strains (along the x, y axis z axis) and six shear strains (xy, xz, yz, zx and zy). Some of these strain components are in figure 7. Defining these nine strain components defines the deformation of a 3D object completely.

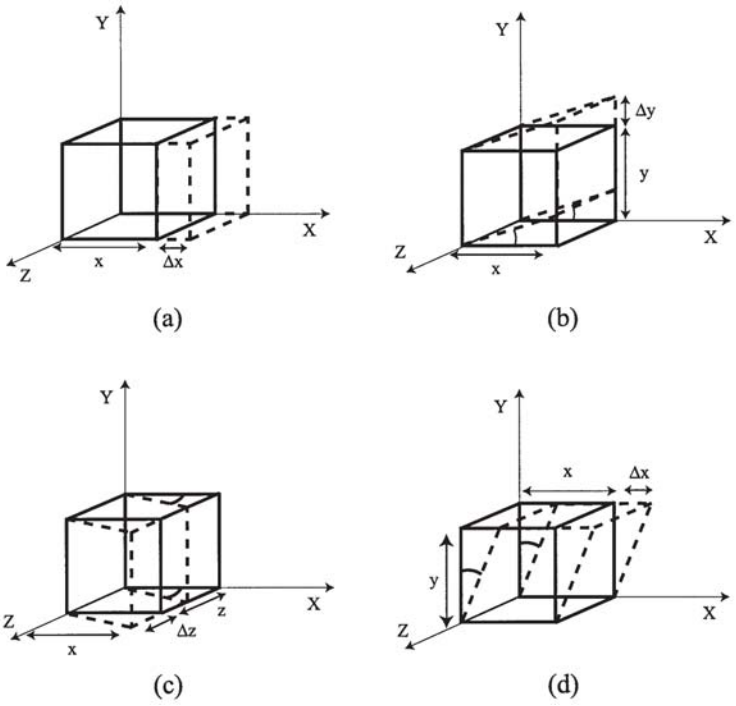

Figure 7: Deformation of a 3D object is described by three normal and six shear strain components. One normal component (a) and three shear components [**ε**yx (b), **ε**zx (c) and **ε**xy (d)] are illustrated here

Ref: Eur J Echocardiography, 2000; 1:157 [74]

STRAIN RATE

Strain Rate is the speed at which deformation occurs. It is represented by symbol ε' and has the unit per sec. In most general situation, strain does not necessarily have a cyclic nature, as one can only deform an object once.

Since a 3D object has nine strain components all occurring at a specific rate, nine Strain Rates can be defined

RELATIONSHIP BETWEEN STRAIN AND WALL THICKENING

For the heart wall thickening is defined as

$$WT = \frac{T_{ES} - T_{ED}}{T_{ED}} \quad \ldots\ldots\ldots 2^{74}$$

with T_{ES} and T_{ED}, end systolic and end diastolic wall thickness respectively. This expression is clearly identical to equation 1. In other wards wall thickening is nothing but myocardial strain measured in one dimension. Rate at which the myocardial wall thickens and thins is nothing but the one dimensional strain rate.

CO- ORDINATE SYSTEMS

In order to uniquely define different positions in space, a 3D co-ordinate system must be constructed. This is defined to be a set of three different, mutually non co-planar unit vectors having the same origin. The position of any point within the concomitant 3D space can be expressed relative to these unit vectors. In this way, every single spatial point within the co-ordinate system can be ascribed a unique co-ordinate.

Similarly, the Strain component measured will depend on the co-ordinate system used. Using the most appropriate co-ordinate system is important, as this will facilitate interpretation of the measurements and will reduce mathematical complexity required to describe the deformation. The possible co-ordinate systems which can be used in ultrasonic cardiac strain imaging are as follows

The Heart Coordinate system:

Rather than defining a global Cartesian co-ordinate system in which the whole heart or left ventricle is described (figure 8), a local heart co-ordinate system can be defined. For each point to be interrogated in any myocardial wall, three mutually perpendicular axes can be defined.

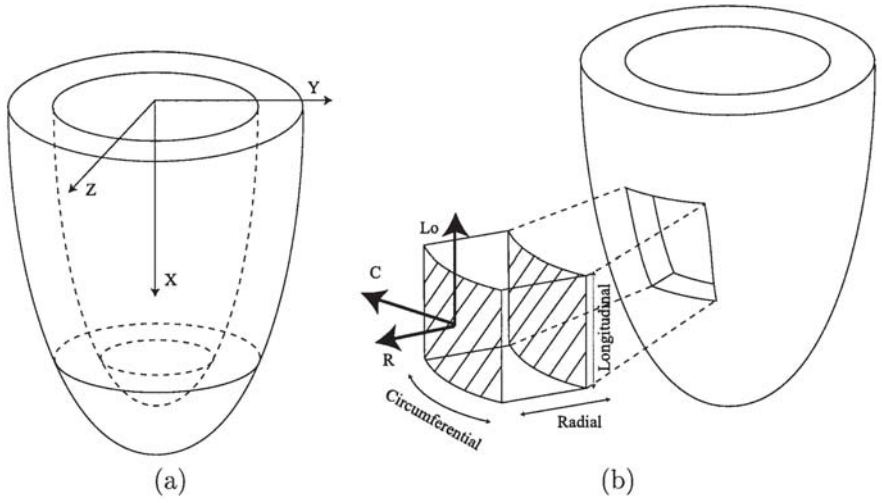

Figure 8: Either a global Cartesian coordinate system (a) or a local heart coordinate system (b) can be defined. However, the latter coordinate system facilitates the physical interpretation of strain measurements of the heart
Ref: Eur J Echocardiography, 2000; 1:158 [74]

The radial (R) axis: Perpendicular to the epicardium, pointing outwards, that is away from the cavity.

The longitudinal (La) axis: Perpendicular to the radial axis, that is, tangent to the epicardium, and pointing towards the base of the ventricle, away from the apex

The Circumferential (C) axis: Perpendicular to both the radial and longitudinal axis, defined in such a way that R-La-C coordinate system is right handed (if one looks along the axis, the rotation from the R to the La-axis is clockwise). Thus the

circumferential axis is directed anti clockwise around the classical echo short axis image. This local co-ordinate system is illustrated in figure 8.

MEASUREMENT OF CARDIAC STRAIN

Since the beginning of the 1980 methods have been proposed to estimate strain by means of ultrasound [73-76]. Originally, most methods were developed for static organs such as liver, breast, kidney or prostate. In these organs, stiffer tissue will deform less than more elastic tissues under the influence of an identical force. Thus in this situation Strain estimation can be directly related to the elastic properties of the tissue. For this reason technique has been termed as elastography.

A series of ultrasound techniques have also been developed to measure myocardial strain in vivo. Several different Strain estimation techniques have been proposed based on the principles of elastography [77,78]. The main difficultly in elastographic strain estimation of heart is the fact that most myocardial segments exhibit a combination of relatively large motion & deformation. Therefore, another approach based on Doppler myocardial imaging principles has been proposed [79]. According to this method strain rate can be expressed as the difference in velocities at both ends of an object. Since local instantaneous myocardial velocities can be measured by color Doppler, this means that strain information can be extracted from real time, digitally stored myocardial data sets

by post processing. One major criticism of velocity, Strain Rate and Strain imaging has been that these techniques are angle dependent, since only the axial component of the true 3D velocity vector or deformation is measured. This problem can partially be avoided by making sure that insonation is either perpendicular or parallel to the myocardial wall during acquisition of the data sets.

Thus taking together Stain Rate and Strain imaging are potentially new tools for the quantification of regional myocardial function. However, clinical relevance of the technique is still under evaluation.

MEASUREMENT OF STRAIN AND STRAIN RATE BY ECHOCARDIOGRAPHY

The echocardiographic measurement of myocardial strain offers a series of regional and global parameters that may be useful is assessment of systolic and diastolic function. Myocardial strain may be measured using a variety of echocardiographic techniques. Although M-mode techniques provide both accurate temporal and accurate spatial resolution, and may therefore be used to measure strain in single dimension, the current era of myocardial strain measurement began with the measurement of strain rate from comparison of adjacent tissue velocities by Heimdal et al[80].

Tissue Doppler Based Strain – Technical Aspects

The velocity of movement of myocardium can be recorded by Tissue Doppler techniques and displayed as a parametric color imaging in which each

pixed represents the velocity relative to the transducer. These data may be expressed graphically as the velocity of the myocardium relative to time (on the x axis).

Rather than examining the motion of a segment relative to the transducer, which is susceptible to tethering to adjacent tissue, myocardial motion may be measured relative to the adjacent myocardium. The instantaneous gradient of velocity along a sample length may be quantified by performing a regression calculation between the velocity data from adjacent sites along the scan line, and these instantaneous data may then be combined to generate a Strain rate curve[11,74]

However velocity regression technique has a few potential pits falls. First, the comparison of adjacent velocities is exquisitely sensitive to signal noise, and the quality of Strain Rate curves may vary depending on the care used in obtaining the underlying velocity data. Optimizing the velocity signal should include avoidance of reverberation artifact and ensuing adequate frame rate (>100 frames/sec). Improvements to the velocity signal by use of harmonic imaging as well as both temporal and spatial averaging are important in optimizing the Strain Rate signal, although this comes at the cost of reducing spatial resolution [81]

Second limitation relates to the limits on spatial resolution that are impaired by imaging at high temporal resolution. If the number of Doppler interrogating beams is limited in an effort to maximize temporal resolution, spatial resolution may be compromised. This may contaminate myocardial velocity signals with

adjacent Left Ventricular blood pool velocities, which are an important source of noise. In turn this will compromise the Strain rate signals. Tracking the sample throughout the cardiac cycle is also important to ensure that sample remains within the myocardium.

Third, like all Doppler techniques, Tissue Velocity based Strain is sensitive to alignment.

Fourth, the derivation of data along the scan line means that the velocity regression technique is unidirectional. Even when tracking is used to try to maintain the sample volume within segment of myocardium, it needs to be kept in mind that myocardium undergoes a wringing, torsional motion so that the sample will inevitably move out of the scanning field in the course of cardiac cycle. This motion has little effect on systolic measurements, because peak Strain Rate occurs early in systole, but it may become important in the measurement of diastolic phenomenon.

Finally, angle changes during the cardiac cycle and with respiratory movement may contribute to drifting of the Strain curve.

These technical challenges of tissue velocity, based strain rate measurements can be avoided by careful acquisition.

Despite these limitations, it is important to acknowledge that this technique has been extensively validated, initially with sonomicormetry[82] and subsequently studies have confirmed correlation with magnetic resonance imaging[83].

POTENTIAL CLINICAL APPLICATIONS OF STRAIN RATE IMAGING

Left Ventricular Function

From animal experiments, the regional Strain values have been validated to correlate with those obtained from sonomicrometry in acute coronary ischemia[82]. In canine models, reduced systolic strain appears earlier and therefore is more sensitive than Doppler Tissue velocity abnormality and semi-quantitative visual wall motion score in acute ischemia[84]. Moreover, the radial peak systolic strain of myocardium correlates linearly with M-mode ejection fraction calculated with the Teichholz equation[85].

The longitudinal systolic strain rate has been shown to correlate linearly with maximal value of the first Left Ventricular pressure time derivative and also peak elastance, which are both global measures of Left Ventricular systolic function and contractility [86,87]. Furthermore, in both normal human and stunned porcine myocardium, the dobutamine induced increase in systolic strain rate preceded the increase in strain itself and also in Left Ventricular systolic wall thickening.[87,88].

Clinically the real time color Strain Rate imaging was shown to moderately correlate which wall motion score of standard echocardiography in 15 patients with acute myocardial infarction[89]. Furthermore, systolic Strain Rate rather than corresponding Doppler Tissue velocity correctly identifies small basal septal infarct induced after percutaneous septal ablation for obstructive hypertrophic

cardiomyopathy, suggesting regional Strain Rate measurement reflects local segmental contractility and is free form adjacent tissue tethering or overall parallel motion of the heart [90].

Stress Echo

Responses of Strain and Strain Rate to stress have been extensively studied in animal models. In normal myocardium, increasing doses of dobutamine are associated with increasing Strain Rate throughout the study, but in contrast, myocardial strain initially increases then decreases as heart rate increases[91]. These changes have been used to argue that Strain Rate is the preferred parameter for the assessment of myocardial function during stress-although they do not account for the greater technical challenges of measuring Strain Rate during stress, nor the degree of differences that occurred in Strain measurements.

Experimental models suggested systolic radial Strain and Strain Rate could clearly differentiate chronic nontransmural from transmural MI during dobutamine stress echocardiography. The transmural extension of the scar could be defined by regional deformation response and correlated closely with baseline regional radial systolic Strain[92].

Valvular Heart Disease

Measurement of myocardial function may be important in understanding the physiological impact of valvular heart disease. Studies in percutaneous heart valve replacement have shown dramatic improvements of Left Ventricular Strain Rate and Strain[93]. Subclinical myocardial dysfunction may be identified as a potential guide to the timing of surgical intervention in regurgitant valve lesions [94].

Right Ventricular Function

Measurement of Right Ventricular Strain and Strain Rate, although feasible[95] and certainly of potential interest in the evaluation of congenital heart diseases, remain challenging. The tissue Doppler approach to radial strain measurement is difficult because the Right Ventricular wall is too thin to permit an adequate regression distance and the place of 2D Strain is undefined in this respect. Strain assessment of septum is complicated because of Right and Left Ventricular components .So the long axis assessment of Right Ventricular function is best performed in the free wall, using apical imaging.

REGIONAL LEFT ATRIAL FUNCTION BY TISSUE DOPPLER VELOCITY AND STRAIN IMAGING

Tissue Doppler imaging quantified regional tissue motion velocity and Strain & Strain rate represent the extent of local tissue deformation and its rate respectively. These novel technologies have been validated for the assessment

of global and regional Left Ventricular function and have also been applied to the evaluation of regional Left Atrial function. From an electromechanical perspective, echocardiographic parameters that assess Left Atrial mechanical function may provide a greater understanding of atrial performance and its relationship with ventricular function.

Assessment of Left Atrial and Appendage Function by Tissue Doppler Imaging.

Spectral pulse Tissue Doppler Imaging and two dimensional color-coded Tissue Doppler Imaging can be applied to generate a myocardial velocity curve to assess a regional Left Atrial function, by placing a small volume at an atrial segment of interest. Usually a sample volume of about 2 mm for measuring velocity and not more than 12mm of length for Strain and Strain Rate is preferred because of thin Left Atrial wall. Spectral pulse Tissue Doppler Imaging that has a better temporal resolution however can measure only one segment at time, whereas color coded Tissue Doppler images can be processed offline and offer simultaneous multi segment analyses of velocities and other Tissue Doppler derived parameters, such as Strain and Strain Rate. Thus, different Left Atrial walls with their corresponding levels from the Mitral annulus can be compared and assessed, such as septal and lateral walls in a apical for chamber view, anterior and inferior walls in a two chamber view [54, 96-99]

The atrial myocardial velocity curve consists of three major deflections, ventricular systolic (Sa), early ventricular diastolic (Ea), and late ventricular diastolic (atrial contraction Aa) waves (figure 9).

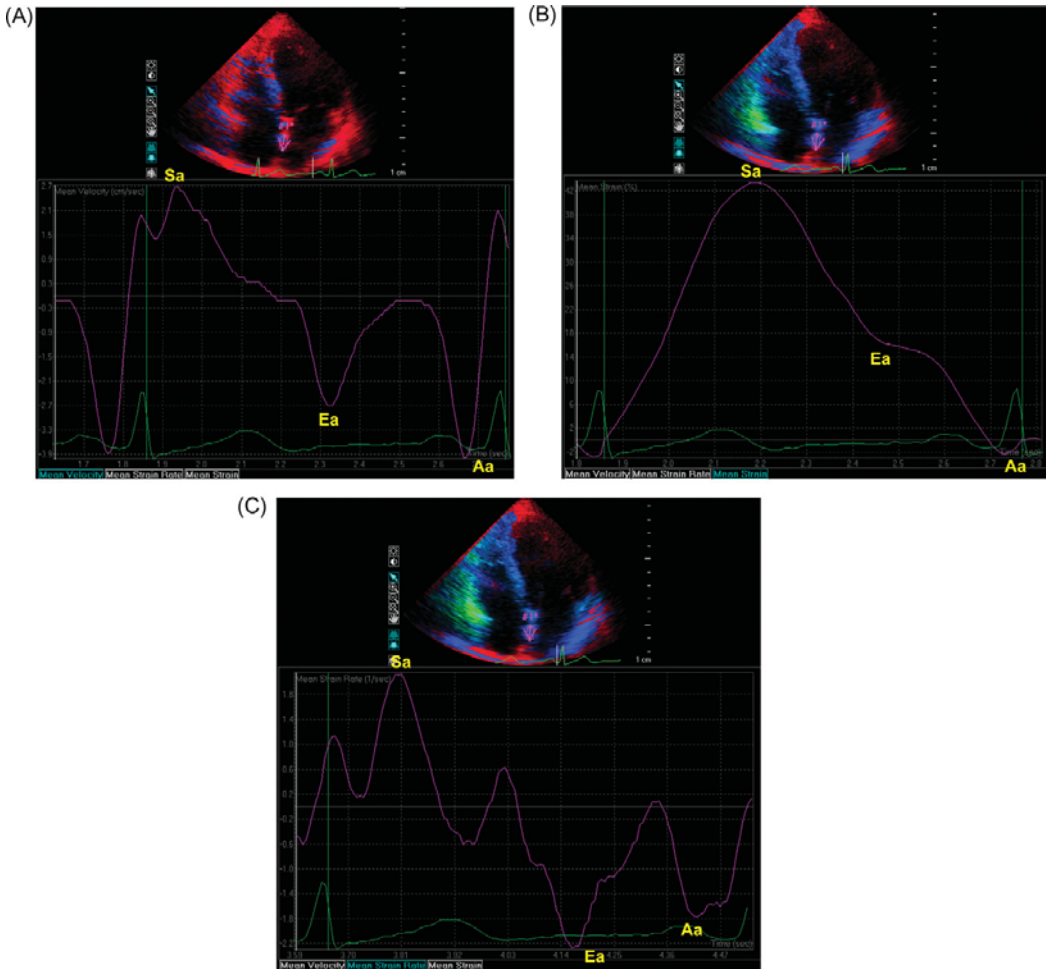

Figure 9: Assessment of regional atrial myocardial function by tissue Doppler imaging. The sample volume was placed at the mid-level of inter-atrial septum on color tissue Doppler imaging image to reconstitute the myocardial velocity (A), strain (B), and strain rate (C) curves. They consist of ventricular systolic (Sa-wave), ventricular early diastolic (Ea-wave), and late diastolic (atrial contraction, Aa-wave) components
Ref: Europace (2008) 10, iii63 [100]

In addition, the three components in Strain and Strain rate imaging can also readily identified (figure 9). The Aa wave has been regarded as a direct measure of regional active atrial contraction on the longitudinal axis, which might be less load dependant [54,55,96,97]. The Sa and Ea waves may represent the

passive expansion and emptying components of the Left Atrial function [55,56,99] The feasibility and reproducibility of Tissue Doppler parameters, in particular the peak velocity (VAa) and peak Strain Rate (SRAa) of the active atrial contraction, have been demonstrated in previous studies, in which both the inter and intra observer variability for measuring the VAa were reported to be within 10% [54,96,99]. Although the velocity data could be easily obtained in nearly all patients, strain rate measurements were only feasible in about 95% of patients due to relatively higher noise signal ratio[98].

Left atrial appendage (LAA) is highly contractile structure with a pattern of contractions totally different from that of the Left Atrial main body. It is more complaint and therefore plays an important role in Left Atrial reservoir function, especially during increase in LA pressure and volume[101]. Transeophageal echocardiography (TEE) provides essential information about LAA structure and function. In addition to the LAA size, emptying and filling velocities[101,102] a characteristic triphasic Tissue Doppler profile can be obtained readily at the tip, the septal or lateral wall of the LAA by TEE.

Atrial Pump Function and Electromechanical Coupling in Healthy Subjects

In healthy subjects, the right atrial (RA) free wall has been found to have the highest VAa or SRAa [96,97]. Zhang et al studied 131 healthy adults aged from 22 – 31 years, measured the atrial wall VAa at mid level. This was found to be significantly higher in the Right and left atrial free wall[97]. The difference in the

atrial velocities at different sites was attributed to an atrial free wall motion higher than that of bounded Inter atrial septum. Furthermore, the larger pectinate muscle in the Right Atrium can perhaps generate a more pronounced and sustained longitudinal movement in the relatively low pressure system of the right ventricle. In the same study when older ($\geq$ 60 y) and younger (<60 yr) age groups were compared VAa was elevated in both Right and Left Atrial free wall in the older age group. Thomas et al [53] also illustrated the effect of age on atrial pump function in their study of 92 healthy subjects in that the older age group had a significantly higher VAa. With aging there is impairment of Left Ventricular myocardial relaxation and early filling, with subsequent increase in the Left Atrial pressure and volume. By the Franks- Starling law, over stretching the atrial myocardium results in augmentation of Left Atrial contractility [53,103-105]. The results of direct measurement of the Left Atrial mechanical function by Tissue Doppler Imaging have corroborated previous observation by Doppler echocardiography that, with aging, there is shift in the relative contribution of the different haemodynamic phases of the Left Atrial function in the Left Ventricular filling, from a reduction of the passive to a compensatory increase in active emptying function [106]

Left Atrial Function by Tissue Doppler and Strain imaging in Atrial Fibrillation

Atrial fibrillation is the most common cardiac arrhythmia associated with an at least two fold increase in morbidity and mortality and occurs in 0.4% of the

general population increasing to 5% in those > 65 years old[107]. With the loss of atrial booster pump function, the Left Atrial –Left Ventricular pressure gradient during early Left Ventricular filling is enhanced by elevation of the Left Atrial pressure to maintain stroke volume[108]. Thus, a reduction in both Left Atrial compliance and volume has been observed with the onset of Atrial Fibrillation that further decreases cardiac function and increased the risks of thrombo-embolism.

Using Tissue Doppler derived Velocity and Strain- rate parameters; it was found that Left Atrial mechanical function was significantly decreased in Atrial Fibrillation as reflected by reduction in the Sa and /or Ea wave in the absence of Aa wave in the late diastole. Wang et al[99] compared 52 patients suffering form Atrial Fibrillation for less than 1 year with 27 matched normal control subjects. By placing the sample volume at basal level of the Left Atrial wall, the velocity measure during ventricular systole and early diastole, as well as the Strain Rate during ventricular systole were markedly reduced in patients with Atrial Fibrillation. In particular, the Strain rate during ventricular early diastole was significantly lower in patients who failed the initial cardioversion or reverted to Atrial Fibrillation within 4 weeks after initial successful cardioversion when compared with those who were successfully cardioverted and remained in sinus rhythm. Similarly, Salvo and colleagues placed the sample volume at the mid Left Atrial walls and obtained myocardial Velocity, Strain and Strain Rate data at both at ventricular systole and early diastole in 65 patients suffering from lone Atrial

Fibrillation for at least 3 months[56]. All measure myocardial properties were significantly reduced in patients with Atrial Fibrillation when compared with normal subjects. Furthermore the Strain Rate during ventricular systole from the individual Left Atrial wall was much lower in patients who had more than one recurrent Atrial Fibrillation episode with a 9 month follow-up period after cardioversion than those in sinus rhythm. These results may reflect the decreased compliance of Left Atrial wall in patients with Atrial Fibrillation, which is in agreement with several studies demonstrating that during Atrial Fibrillation the reservoir and conduit function are impaired and the booster pump function is lost.

Transthoracic DC cardioversion is one of the most widely used and effective treatments in restoration of sinus rhythm and may ameliorate the detrimental effects of Atrial Fibrillation, as well as prevent the development of associated tachycardia induced cardiomyopathy[109]. However it has high recurrence rate determined by multiple factors, including patient's age, origin and duration of Atrial Fibrillation and functional class and degree of Left Atrial enlargement. Previous studies have demonstrated that severe impairment of atrial deformation during ventricular early diastole was an independent predictor of recurrent Atrial Fibrillation after adjusting for clinical and other echocardiographic parameters[56,99]. Nevertheless, combining Left Atrial mechanical dysfunction with the degree of Left Atrial enlargement gave the strongest predictive value of Atrial Fibrillation recurrence [99].

Atrial stunning is characterized by reduced atrial mechanical function after restoration of sinus rhythm from Atrial Fibrillation which may last several weeks with associated increased thromboembolic risk[110]. Thomas et al[57] demonstrated a gradual recovery of atrial pump function after DC cardioversion, by use of strain imaging in 37 patients with chronic Atrial Fibrillation who had a restored sinus rhythm.

Left Atrial Function by Tissue Doppler and Strain imaging In Ischemic Heart Disease

Atrial contractile dysfunction appears early in ischemic heart disease irrespective of previous myocardial infarction, co-existing systolic dysfunction or severity of diastolic dysfunction. Yu et al[96] found that the VAa measured at mid level of the Interatrial septum and the Left Atrial lateral wall in the apical four chamber view were drastically reduced in 118 patients with ischemic heart disease when compared with 100 normal subjects . A poor Left Ventricular ejection fraction and the presence of a restrictive Left ventricular filling pattern were the most important determinants of Left Atrial contractile dysfunction in ischemic heart disease. The dramatic reduction of Left Atrial VAa in the presence of a restrictive filling pattern suggested the presence of possible Left Atrial contractile dysfunction, which has been proposed in these patients.

Left Atrial Function Tissue Doppler and Strain Imaging in Advanced Heart Failure and Cardiac Resynchronization Therapy

The atria adapt to changes in ventricular filling commonly observed in congestive heart failure by adjusting the relative proportion of reservoir, conduit and pump components in order to maintain the ventricular stroke volume. However depression of atrial pump performance will eventually occur as the heart failure progresses, despite increased atrial preload due to myopathic process and/or over distention of the atrial fibers[111]. Thus, in chronic heart failure, both the velocity and Strain during Left Atrial contraction are attenuated. However, the Left Atrial mechanical function can be modified by heart failure treatment, such as cardiac resynchronization therapy (CRT), which is of proven benefit to the advanced heart failure patients with prolonged QRS duration. In this regard, Tissue Doppler Imaging helps investigate the improvement in regional Left Atrial mechanical function after CRT .Yu et al[15] examined atrial function and remodeling in the population by a combined assessment using conventional and new echocardiographic imaging tool. It was observed that atrial remodeling was evident at 3 months after CRT, identified by reduction in the atrial area and volume before and after atrial systole. Meanwhile, atrial contraction function was significantly improved, including increase in atrial emptying fraction, peak velocity and Strain during atrial contraction. There was also improvement of Left Atrial peak Strain during ventricular systole and early diastole, which signifies an improvement in atrial compliance. These changes were mainly observed in responders showing LV remodeling

Left Atrial Function in Mitral Stenosis

Due to inflow obstruction, the atrial booster pump contributes less to Left Ventricular filling in Mitral Stenosis even during sinus rhythm, despite a proportional increase, with increasing severity, in the Left Atrial pre load [112]. Left atrial function in Mitral Stenosis has not been studied too extensively in the past. Sato S et al [113] demonstrated that the impaired atrial reservoir and pump function are associated with a reduction in Left Atrial compliance and intrinsic myocardial contractility. Stefanadis et al [114] assessed Left Atrial function in Mitral Stenosis by pressure area relation in 15 patients with Mitral Stenosis. Patients with Mitral Stenosis were found to have increased Left Atrial size and decreased Left Atrial pump function, as indicated by the decreased Left Atrial systolic emptying index and the decreased Left Atrial stroke work index. After Balloon Mitral Valvotomy there was significant increase in the Left Atrial A loop in patients with sinus rhythm and in the Left Atrial V loop in patients with atrial fibrillation. In Mitral Stenosis, the increased Left Atrial stiffness before valvuloplasty returned to normal immediately after the procedure. Mi-Seung Shin et al [115] evaluated Left Atrial Tissue Doppler velocities and volumes by Tissue Doppler imaging and 3-D echocardiography in patients with Mitral valve disease. They found lower regional peak systolic and late diastolic left atrial tissue velocities in 22 patients with moderate or severe Mitral Stenosis and also a lower Left Atrial ejection fraction in Mitral Stenosis patients as compared to control population. P Casto et al[116] assessed atrial reservoir function by Strain imaging in asymptomatic patients with

mild to moderate Mitral Stenosis. They found that the atrial reservoir function was abnormal and the degree of impairment was found to predict cardiovascular events at 3 years

MATERIALS AND METHODS

Study Design:

This was a prospective descriptive trial performed over 12 months from Dec 2008 to Dec 2009

Setting:

CMC Vellore is a 2000 bedded Tertiary care teaching hospital. Patients were recruited from outpatient department and those admitted with severe Mitral Stenosis. Twenty five patients with severe Mitral Stenosis in sinus rhythm and 25 ages matched Controls were enrolled in the study.

Subjects:

Inclusion Criteria

Control Group: Patients with no cardiovascular risks factors and not on any cardiac medications.

Mitral Stenosis Group: Patients with isolated severe Mitral Stenosis (with no other significant valvular lesions) in sinus rhythm, planned for Balloon Mitral Valvotomy.

Exclusion Criteria

1. Patients of Mitral Stenosis not suitable for Balloon Mitral Valvotomy
2. Patients with Atrial Fibrillation
3. Patients with more than mild Aortic or Mitral regurgitation (pre or post Balloon Mitral Valvotomy)
4. Patients undergoing Balloon Mitral Valvotomy as an emergency procedure
5. Patients who had undergone Closed Mitral Valvotomy or Balloon Mitral Valvotomy or any form of cardiac surgery in the past.
6. Patients with Coronary Artery Disease, Hypertension & Diabetes Mellitus
7. Patients with poor echo windows or incomplete study..

Clinical Assessment

All patients were subjected to thorough history taking, full clinical examination, 12 lead ECG, full 2D, M mode & Doppler transthoracic echocardiographic study in standard precordial views. Left Atrial regional function and deformation properties were studied using Tissue Doppler Velocities, Strain and Strain Rate imaging. All details were plotted in tables and statistically studied

Echocardiographic And Doppler Studies

Each individual included in the study underwent standard transthoracic echocardiogram and Tissue Doppler imaging in left lateral decubitus position in expiratory apnea by 5 Hz probe on iE33 (Philips Medical system ,Andover ,Massachusetts ,USA). All the measurements of ventricular parameters were

recorded using the leading edge technique and in accordance with the recommendations of American Society of Echocardiography[117,118]. Following parameters were obtained from the M mode guided pictures in parasternal long axis view ; Left Atrial dimensions in mm ,Left Ventricular internal dimensions in both systole and diastole (LVIDDs and LVIDDd) in mm, thickness of interventricular septum (IVSd) and posterior wall (PWTd) at end diastole in mm. LVEF was calculated by modified Simpson's method[117]. MVA was calculated by planimetry and Pressure Half Time[119]. Color flow Doppler was used to detect presence of valvular regurgitation.

Left atrial volume and size was measured by 2D echocardiogram in apical 4 chamber view and Ejection Fraction was calculated using modified Simpson's method. Maximum Left Atrial volume was measured during ventricular systole when mitral valve was closed and smallest Left Atrial volume during ventricular late diastole with pulmonary veins and mitral apparatus excluded from volume measurement [31] (see figure10)

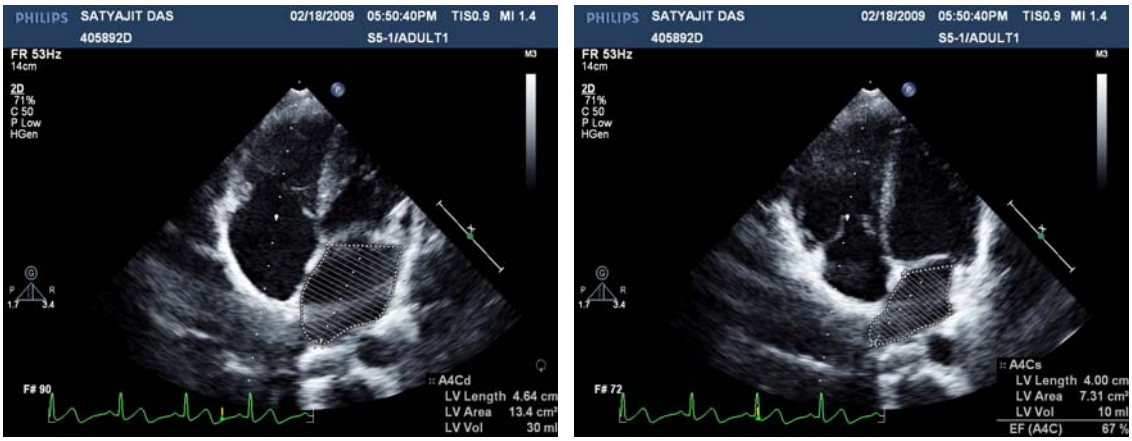

Figure:10 Left atrial maximum and minimum volume measurement in A4C in control

Pulse Waved Tissue Doppler Imaging

Tissue Doppler imaging was performed in apical four chamber view by placing the sample volume at mid point of Interatrial Septum and Lateral left atrial wall. Peak early diastolic velocity (E') and late diastolic velocity (A') were recorded. A high frame rate (>110 frame/sec) was selected for the study. Special case was taken for correct alignment of the Doppler beam parallel to Interatrial septum. Doppler measurements were obtained during end expiration. An appropriate velocity scale was chosen to avoid data aliasing.

Atrial Strain and Strain Rate Imaging

Color Doppler myocardial images were acquired using a narrow sector (usually $\leq$ 30 degree) to attain a frame rate >110 frames/sec. Attempts were made to align the atrial wall parallel to the Doppler beam. Because of the thin atrial walls, a narrow (10X2.5mm) sample volume was selected[57]. Images were acquired followed by offline strain and strain rate evaluation using QLAB software. Sample volume was placed at mid Interatrial septum (fig 11a & 11b) and mid Lateral wall of Left atrium (fig 12a & 12b) in apical four chamber view [15]. Mean Strain (Strain and Strain Rate) parameters were recorded at end diastole defined as peak of R wave in ECG and end systole defined as the end of T wave in ECG [56].

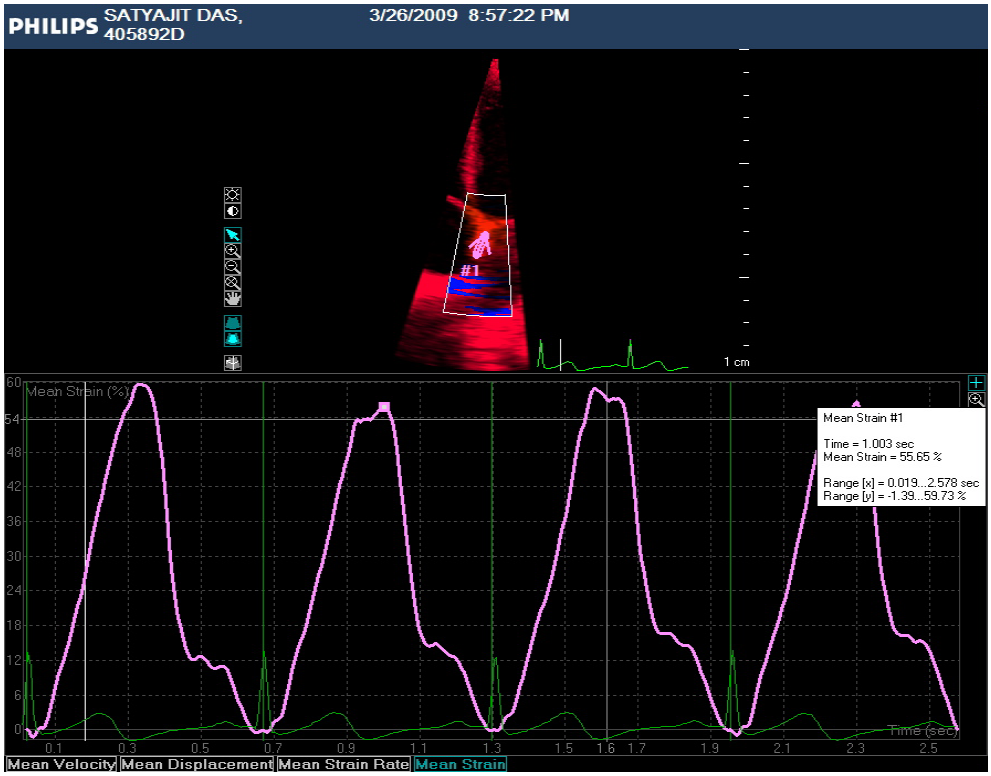

Figure13 a: Strain curve with sample volume at mid point of inter atrial septum

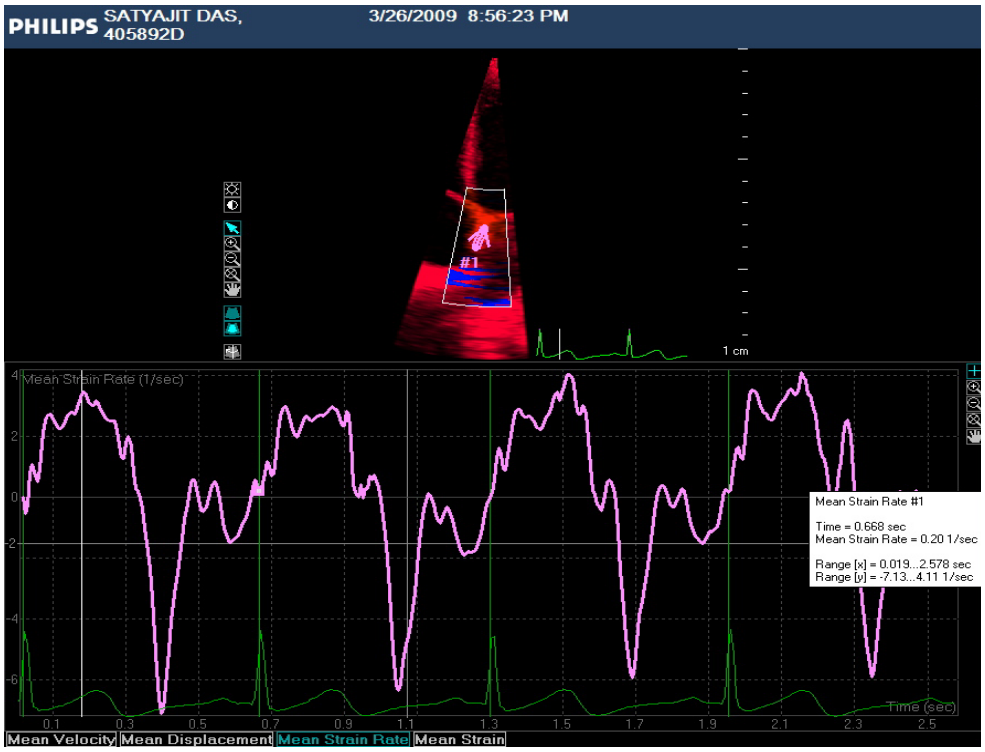

Figure 13b: Strain Rate curve with sample volume at mid point of interatrial septum

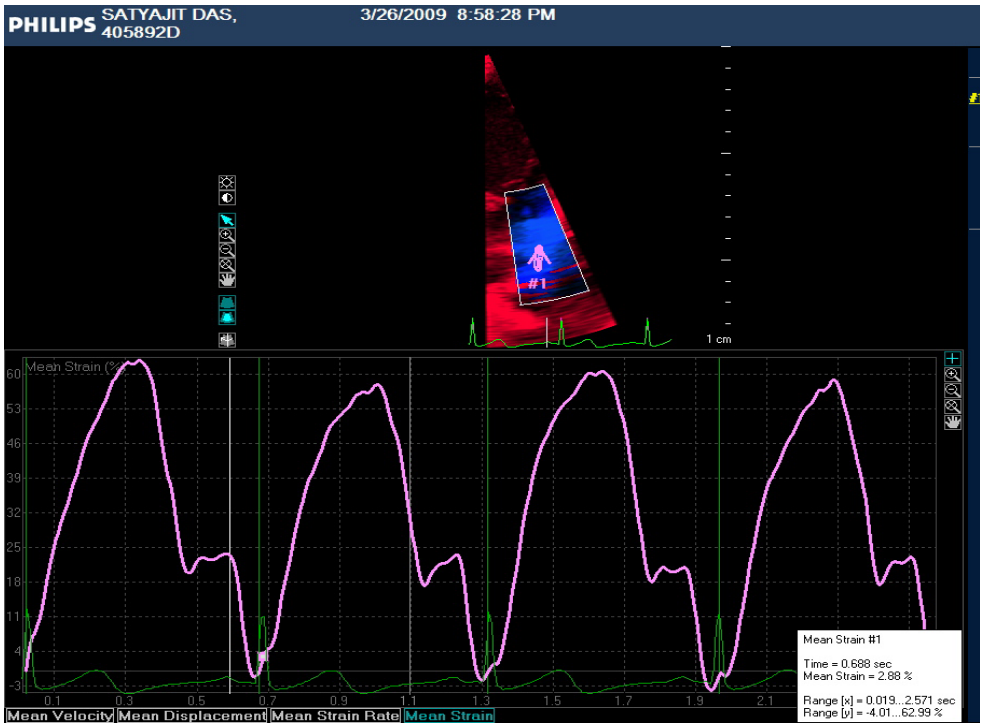

Figure 14a: Strain curve with sample volume at mid point of lateral LA wall

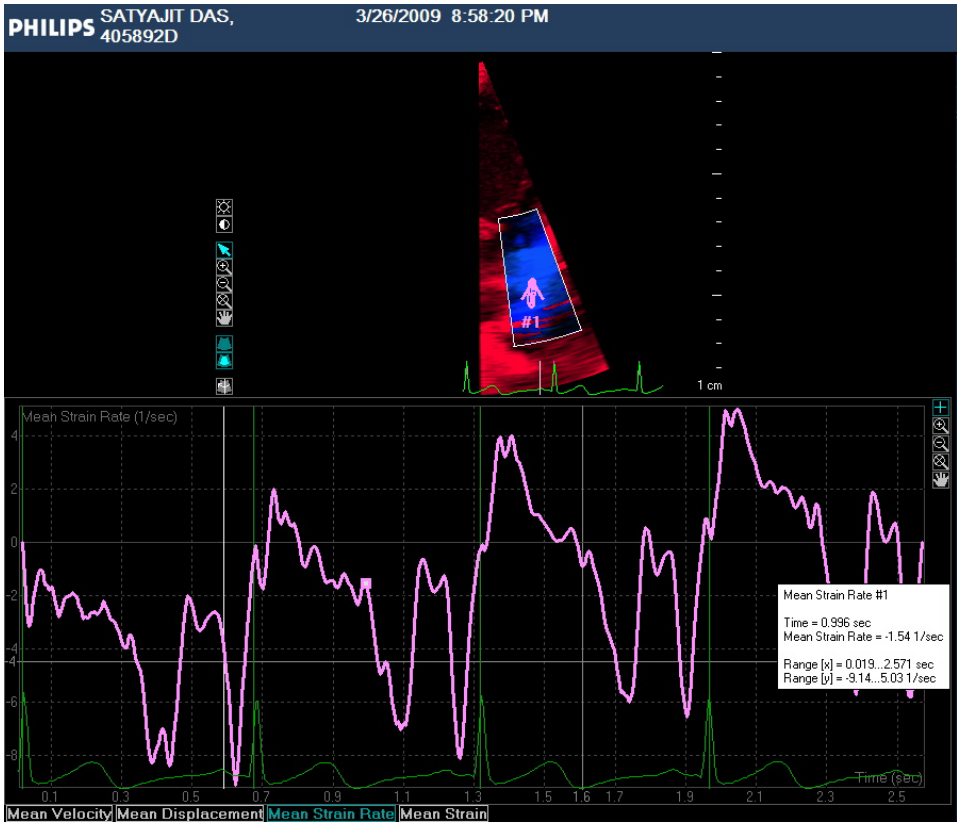

Figure14b: Strain Rate curve with sample volume at mid point of lateral LA wall

Balloon Mitral Valvotomy and Follow Up

All patients of Mitral Stenosis underwent Balloon Mitral Valvotomy through trans-septal single balloon technique. Balloon Mitral Valvotomy was considered successful when post procedure echo revealed mitral valve area $\geq$ 1.5cm2 by echo with less then 2+ Mitral Regurgitation[120]. Tissue Doppler velocities, Strain and Strain Rate parameters were measured 24 hours after Balloon Mitral Valvotomy in manner similar to pre procedure evaluation.

Statistical Analysis: All statistical analysis were performed by SPSS for Windows 16.0 (Chicago, USA) and RGui 2.8.0. Numerical results were expressed as Mean ± SD. Comparisons between Cases and Controls were done using the student's t test and Mann- Whitney U test for independent samples. Pre and Post Balloon Mitral Valvotomy analysis were done using the Paired Sample t test and Wilcoxon Signed Rank test. The level of significance is 5% (p value< 0.05). Pearson Correlation coefficient is used to find the correlation between the two variables.

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RESULTS

During the period of 1 year, 50 patients were included in the study of which 25 were of severe Mitral Stenosis (2D MVA= 0.87 ± 0.14 cm^2) and 25 were age matched Controls (28.7 ± 6.8 vs 30.7 ± 8.9 years; p= 0.34: MS vs Control). Left ventricular systolic and diastolic dimensions and Left Ventricular Ejection Fraction were comparable in both the groups (Table 1).

Table 1: Baseline Characteristics

Variable	Controls (n=25)	Mitral stenosis (n=25)	p value
Age of the Subject	30.7 ± 8.9	28.7 ± 6.8	p = 0.38
M mode LV dimensions in Diastole (mm)	41.9 ± 3.6	39.7 ± 5.9	p = 0.14
M mode LV dimensions in Systole (mm)	27.9 ± 2.5	26.8 ± 3.9	p = 0.21
M mode Posterior wall Dimensions (mm)	9.2 ± 1.3	9.2 ± 1.0	p = 0.89
M mode Interrventricular Septum Dimensions (mm)	9.3 ± 1.1	9.4 ± 1.1	p = 0.79
LV EF by Simpson's method (%)	61.0 ± 3.2	62.4 ± 3.9	p = 0.17
Peak TR Gradient (mm Hg)	17.2 ± 4.9 (n=18)	55.0 ± 32.4 (n=25)	p = <0.001
M Mode Right Ventricular Dimension (mm)	9.3 ± 1.1	9.1 ± 1.1	p = 0.76

Various predetermined 2D Left Atrial parameters in Mitral Stenosis patients were compared with healthy Controls. As shown in Table 2, M mode LA dimensions, maximum LA size in A4C, maximum and minimum LA volumes, were significantly higher in patients with mitral stenosis. LA ejection fraction as calculated by modified Simpson's method was lower in Mitral Stenosis patients when compared to controls (23.4 ± 6.7 % vs 58.3 ± 6.6 %;p<0.001 ;MS vs Control).

Table 2: Left Atrial Echo parameters

Variable	Controls (n=25)	Mitral Stenosis (n=25)	p value
M Mode Left Atrial Dimensions (mm)	29.1 ± 4.2	42.4 ± 7.6	p < 0.001
Maximum LA size in A4C (mm)	45.6 ± 4.7	64.4 ± 8.1	p < 0.001
2 D Left Atrial Maximum Volume (ml)	35.4 ± 10.7	90.6 ± 31.1	p < 0.001
2 D Left Atrial Maximum Volume (ml)	14.8 ± 4.9	69.9 ± 27.3	p < 0.001
Left Atrial EF by Simpson's Method (%)	58.3 ± 6.6	23.4 ± 6.7	p < 0.001

Left Atrial function as assessed by Tissue Doppler Imaging showed significant lower E' and A' Diastolic velocities in Mitral Stenosis when compared to controls (Table 3).

Table 3 : TDI parameters

Variable	Controls (n=25)	Mitral Stenosis (n=25)	p value
IAS Pulse Wave E' Velocity (cm/sec)	9.8 ± 2.7	6.9 ± 3.5	p = 0.002
IAS Pulse Wave A' Velocity (cm/sec)	9.2 ± 2.9	7.1 ± 2.6	p = 0.01
Left Atrial Lateral Wall Pulse Wave E' Velocity (cm/sec)	15.9 ± 4.3	6.6 ± 1.9	p <0.001
Left Atrial Lateral Wall Pulse Wave A' Velocity (cm/sec)	13.1 ± 3.6	7.4 ± 2.9	p <0.001

Strain and Strain Rate imaging of Left Atrium in patients with Mitral Stenosis showed a lower ventricular end Systolic Strain as measured at Atrial Septum (11.4 ± 6.3 % vs 29.6 ± 10.5% ;p<0.001 ;MS vs Control) and Left Atrial Lateral Wall (18.2 ± 8.8 % vs 28.4 ± 14.0% ;p=0.004; MS vs Control). IAS Strain Rate at ventricular end Diastole was lower in patients with Mitral Stenosis when compared with Controls (-0.16 ± 0.35 vs 0.23 ± 0.55; p=0.004). Apart from the above mentioned Strain abnormality all other Systolic and Diastolic Strain and Strain Rate parameters were not significantly different in two groups as shown in Table 4.

Table 4: Strain and Strain Rate parameters in MS as compared to controls

Variable	Controls (n=25)	Mitral Stenosis (n=25)	p value
IAS Strain at Ventricular End Systole (%)	29.6 ± 10.5	11.4 ± 6.3	p<0.001
IAS Strain at Ventricular Late Diastole (%)	-0.04 ± 0.41	0.05 ± 0.33	p = 0.36
IAS Strain Rate at Ventricular End Systole (per sec)	-0.17 ± 1.01	0.15 ± 0.85	p = 0.24
IAS Strain Rate at Ventricular Late Diastole (per sec)	0.24 ± 0.55	-0.16 ± 0.35	p= 0.004
Left Atrial Lateral Wall Strain at Ventricular End Systole (%)	28.4 ± 14.0	18.2 ± 8.8	p= 0.004
Left Atrial Lateral Wall Strain at Ventricular Late Diastole (%)	0.07 ± 0.61	0.01 ± 0.56	p = 0.7
Left Atrial Lateral Wall Strain Rate at Ventricular End Systole (per sec)	0.34 ± 0.71	0.15 ± 0.60	p = 0.3
Left Atrial Lateral Wall Strain Rate at Ventricular Late Diastole (per sec)	0.13 ± 0.4	-0.12 ± 0.49	p=0.055

All patients of Mitral Stenosis included in the study underwent successful BMV with post BMV 2D mean Mitral Valve area of 1.94 ± 0.25 cm^2 by palnimetry. Post BMV LA volumes (maximum and minimum) and M mode LA dimensions reduced significantly as compared to pre BMV as shown in Table 5. However LA size as measured in A4C remained unchanged (64.4 ± 8.1 vs 62.3 ± 7.3 mm ; p =0.06: pre BMV vs post BMV) post procedure.

Table 5: Echo/Doppler parameters Pre and Post BMV

Variable	Pre BMV (n=25)	Post BMV (n=25)	p value
M Mode Left Atrial Dimensions (mm)	42.4 ± 7.6	38.0 ± 6.6	p=0.001
Maximum LA Size in A4C (mm)	64.4±8.1	62.3± 7.3	p=0.064
2 D Left Atrial Maximum Volume (ml)	90.6± 31.1	70.9±26.7	p<0.001
2 D Left Atrial Minimum Volume (ml)	69.8±27.3	48.2±19.8	p<0.001
Left Atrial EF by Simpson's Method (%)	23.4±6.7	32.9±10.0	p<0.001
Mitral Valve Area by 2D (cm2)	0.87±0.14	1.94±0.25	p<0.001
Mitral Valve Area by Doppler (cm2)	0.84±0.15	1.89±0.23	p<0.001
Doppler Peak TR Gradient (mmHg)	55.0±34.4	31.7±15.4	p<0.001

Post BMV Tissue Doppler velocity remained unchanged as measured at interatrial septum however E' velocity at lateral wall improved significantly (6.6 ± 1.9 vs 8.8 ± 2.1; p<0.001:Pre BMV vs Post BMV) as shown in Table 6 and Figure 15a, 15b.

Table 6: Tissue Doppler Velocities Pre and Post BMV

Variables	Pre BMV (n=25)	Post BMV (n=25)	p value
IAS Pulse wave E' Velocity (cm/sec)	6.9 ± 3.5	6.7 ± 2.8	p=0.81
IAS Pulse wave A' Velocity (cm/sec)	7.1 ± 2.6	6.8 ± 2.4	p=0.49
Left Atrial Lateral Wall Pulse Wave E' Velocity (cm/sec)	6.6 ± 1.9	8.8 ± 2.1	p<0.001
Left Atrial Lateral Wall Pulse Wave A' Velocity (cm/sec)	7.4 ± 2.9	7.5 ± 2.4	p=0.82

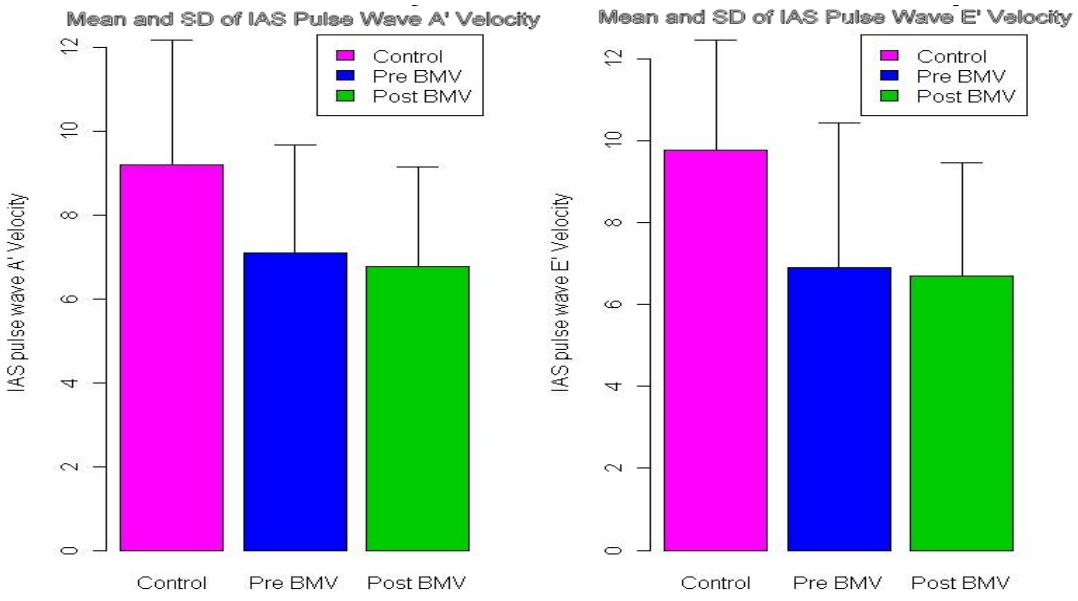

Figure 15a: E' & A' Velocities at interatrial Septum in controls compared with MS pre BMV and post BMV

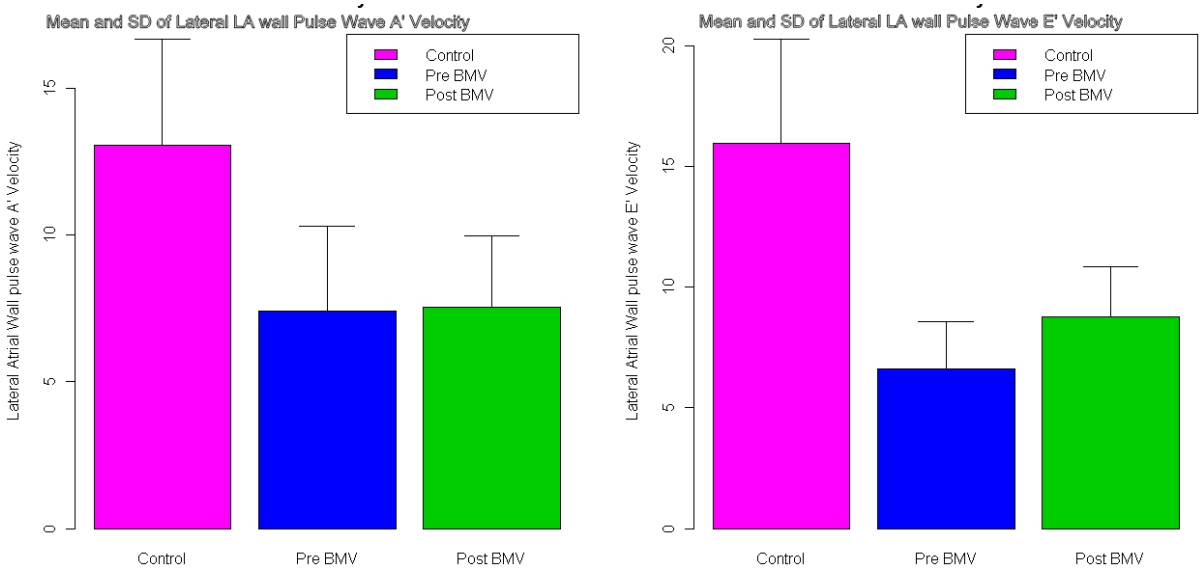

Figure 15a: E' & A' Velocities at Lateral LA wall in controls compared with MS pre BMV and post BMV

Post Balloon Mitral Valvotomy Strain imaging revealed improvement in left atrial IAS systolic Strain (11.4 ± 6.3% vs 18.0 ± 10.6%; p=0.021) as shown graphically in figure 16a and a trend towards improved lateral wall Systolic Strain (18.0 ± 8.8 % vs 22.8 ± 10.6; p= 0.07) (figure 16b).

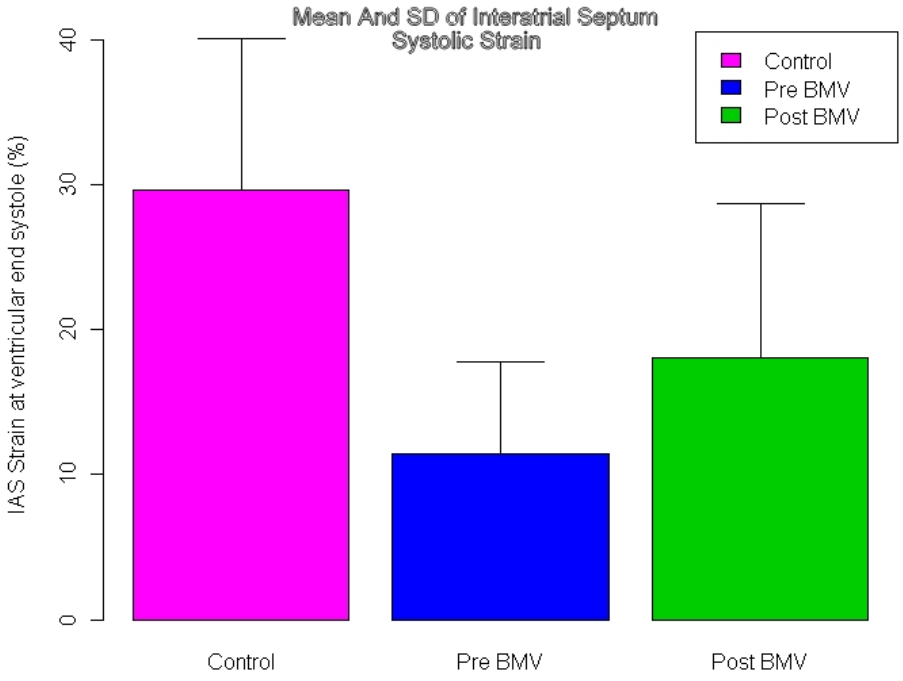

Figure 16a: Atrial Septum Systolic Strain in controls compared with Mitral Stenosis Pre and Post BMV

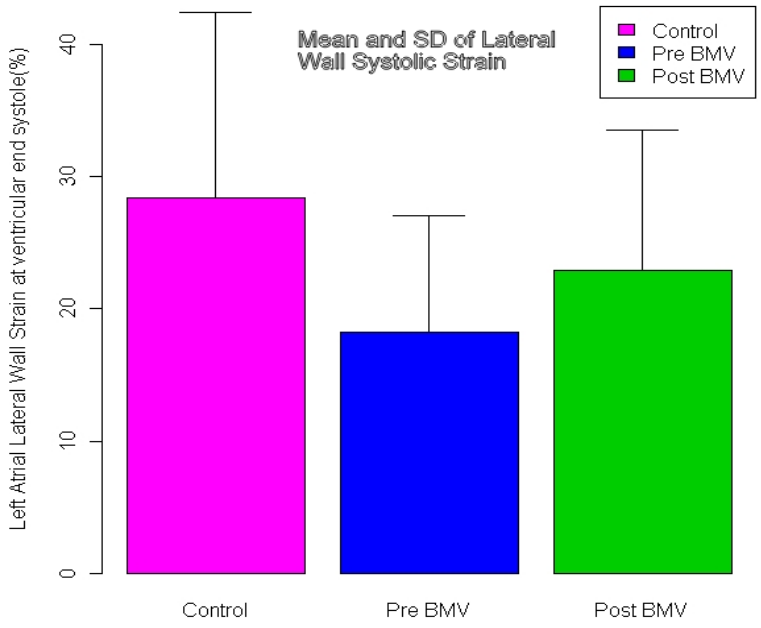

Figure 16b: Left Atrial Lateral Wall Systolic Strain in controls compared with Mitral Stenosis Pre and Post BMV

Atrial lateral wall Strain Rate at end systole was reduced after procedure as compared to pre BMV values. Apart from these all other Systolic as well as Diastolic Strain and Strain Rate parameters were comparable to pre BMV level as shown in Table 7

Table 7: Strain and strain rate parameters Pre and Post BMV

Variables	Pre BMV (n=25)	Post BMV (n=25)	p value
IAS Strain at Ventricular End Systole (%)	11.4 ± 6.3	18.0 ± 10.6	p=0.02
IAS Strain at Ventricular Late Diastole (%)	0.05 ± 0.33	0.13 ± 0.83	p=0.65
IAS Strain Rate at Ventricular End Systole (per sec)	0.15 ± 0.85	0.23±0.49	p=0.70
IAS Strain Rate at Ventricular Late Diastole (per sec)	-0.16 ±0.35	-0.17± 0.68	p=0.89
Left Atrial Lateral Wall Strain at Ventricular End Systole(%)	18.2 ± 8.8	22.8 ± 10.6	p=0.07
Left Atrial Lateral Wall Strain at Ventricular Late Diastole(%)	0.014 ± 0.56	-0.12 ± 1.08	p=0.49
Left Atrial Lateral Wall Strain Rate at Ventricular End Systole (per sec)	0.15 ± 0.60	-0.21 ± 0.63	p=0.02
Left Atrial Lateral Wall Strain Rate at Ventricular Late Diastole (per sec)	-0.12 ± 0.49	0.002 ± 0.58	p=0.43

A significant inverse correlation was found between Left atrial Systolic Strain as measured at atrial septum ($p<0.001$, $R= -0.59$) and left atrial maximum volume (Figure-17). A similar correlation was also found between left atrial Lateral wall Systolic Strain and maximum LA volume ($p<0.001$,$R= -0.50$) (Figure 18)

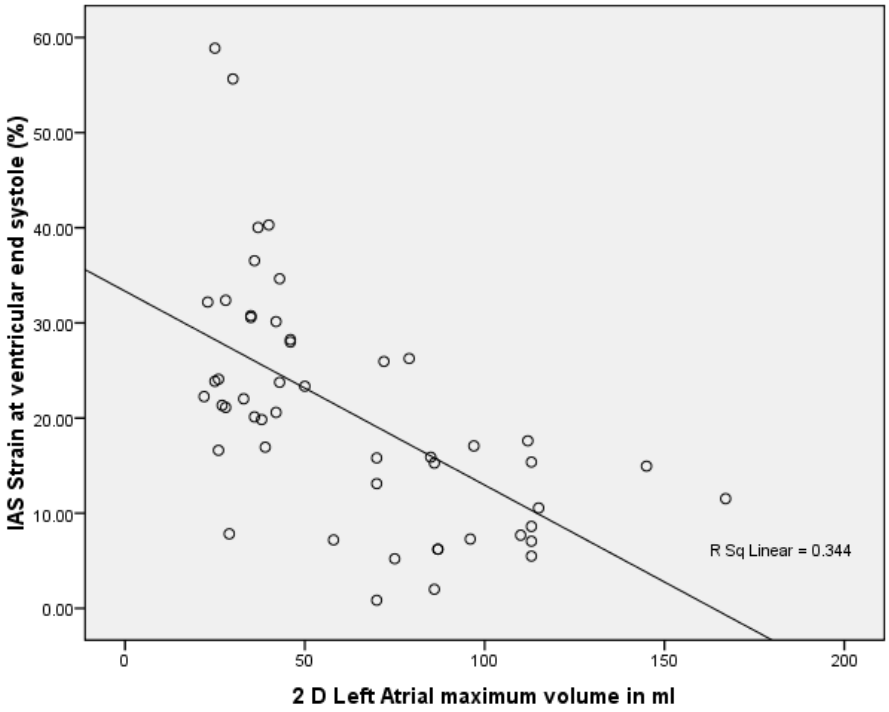

Figure-15: Correlation between IAS Strain (%) and maximal LA volume (ml)

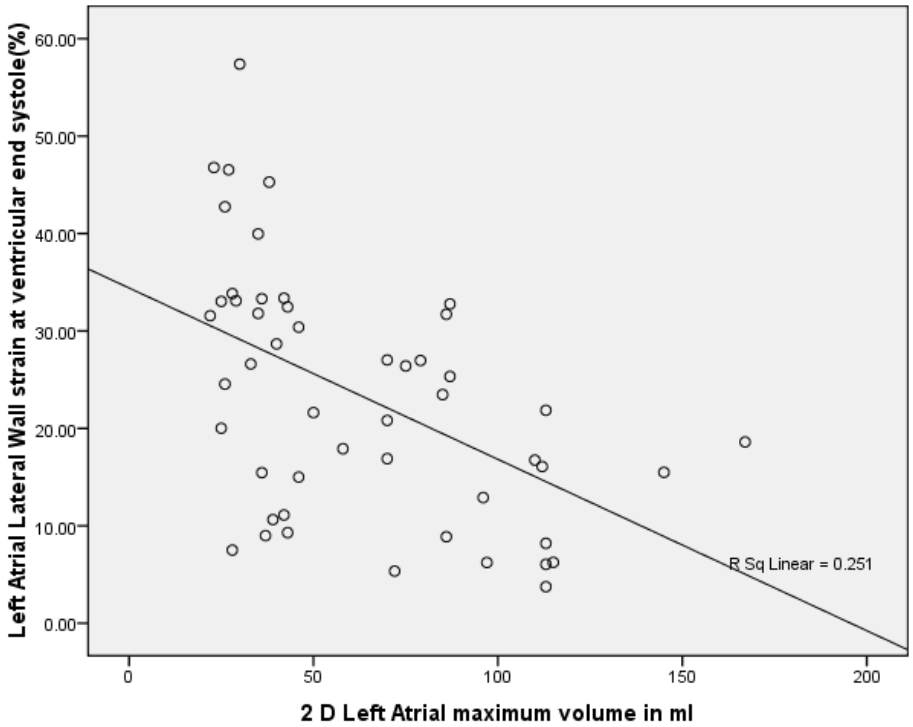

Figure-16:Correlation between Lateral Wall Strain(%) and maximal LA volume (ml)

DISCUSSION

Present study to our knowledge is the first Indian study to assess Left Atrial myocardial deformation properties in patients with severe Mitral Stenosis and effect of Balloon Mitral Valvotomy on Left Atrial function as assessed by Tissue Doppler and Strain imaging. The study demonstrated that Left Atrial Tissue Doppler velocities and myocardial reservoir function assessed by Systolic Strain is abnormal in Mitral Stenosis, with Strain parameters tend to normalize within 24 hours after BMV.

It is known that Left Atrial function is influenced by both atrial and ventricular factors. The atrial factors affecting Left Atrial function include Left Atrial contractility & relaxation, Left Atrial pressure & compliance and rhythm abnormalities. Ventricular factors include Mitral annular displacement, Left Ventricular compliance & relaxation.

Mitral stenosis is one of the many conditions associated with Left Atrial dilatation and remodeling. The atrium enlarges in response to two broad conditions: pressure and volume overload[31,121]. A wide range of Left Atrial pressure exists in rheumatic Mitral Stenosis despite similar Mitral valve area because an important determinant of Left Atrial pressure is LA compliance[122]. Despite similar Mitral Valve area, patients with rheumatic Mitral Stenosis have a different grade of depressed Left Atrial compliance and hence different stiffness.

Abnormal Left Atrial stiffness may induce thrombogenesis, intra atrial stasis with dense spontaneous contrast in Left Atrium and occurrence of Atrial Fibrillation [98].

In our study left atrial size as determined by Left Atrial dimensions in M mode and 2D echo in A4C, Left Atrial maximum and minimum volumes were significantly higher in patients with Mitral Stenosis as compared to Control group. In Mitral Stenosis because of increased resistance at the level of mitral valve there is marked increase in Left Atrial pressure leading to its enlargement. In addition there is increased atrial afterload (at mitral valve level) during atrial contraction, which may lead to decreased Left Atrial pump function as evident in our study by lower Left Atrial ejection fraction in Mitral Stenosis group. Miseung Shin et al [115] has also reported a lower Left Atrial ejection fraction as assessed by 3D echo in 22 patients with moderate to severe Mitral Stenosis when compared with age matched controls. After successful Balloon Mitral Valvotomy with increase in mitral valve area, afterload at mitral valve level decreases leading to improvement in Left Atrial ejection fraction. This was evident as early as 24 hours after Balloon Mitral Valvotomy in our study.

Mitral Stenosis patients are prone to develop atrial arrhythmias. 30-40% of patients with symptomatic Mitral Stenosis are known to develop Atrial Fibrillation [123]. Thus proper assessment of atrial function is particularly important in patients with Mitral Stenosis as it may give an idea about the prognosis. However the previously used methods for assessment of atrial function are either

difficult or inaccurate. M mode from parasternal long axis provides a rapid and easy way that gives an idea about atrial size[32]. It may be inaccurate as it measures Left Atrial dimensions in one plane only however Left Atrial enlargement may not be restricted to that particular plane.

Tissue Doppler imaging with offline assessment of Strain has been introduced for the assessment of atrial reservoir, conduit and contractile function[55]. Left atrial reservoir function is studied by peak systolic value, left atrial conduit function by early diastolic value and left atrial pump function by late diastolic value. This method to assess atrial function in severe Mitral Stenosis has not been evaluated earlier.

Regarding reservoir function, which was assessed in our study by mean Strain at the end systole, as measured at end of T wave was found to be significantly reduced in patients with severe Mitral Stenosis. This was true for both as measured at mid interatrial septum and mid lateral LA wall. There was a significant improvement in Inter Atrial Septal strain within 24 hours after Balloon Mitral Valvotomy and a trend towards improved systolic Strain as measured at lateral LA wall. However Left Atrial size as assessed in A4C remained unchanged at 24 hours after Balloon Mitral Valvotomy. This would suggest that these abnormalities are not related to structural abnormality of left atrial wall per se, if so should have taken longer time to resolve, but are functional abnormality related to degree of Mitral Stenosis.

Di Salvo et al [56] had reported atrial peak Systolic Strain as one of the best predictors for maintenance of sinus rhythm in patients with lone AF. They found patients with recurrence of Atrial Fibrillation after successful cardioversion had lower Systolic Strain as measured at atrial septum and inferior LA wall. Thus improvement in Systolic Strain after Balloon Mitral Valvotomy as found in patients with severe Mitral Stenosis in our study may increase their chance to continue to have sinus rhythm and lower cardiovascular events. Tsang et al [28,30] in more than one study reported that Left Atrial volume was more predictive of future Atrial Fibrillation and other cardiovascular events than Left Atrial dimensions in variable clinical population, suggesting that Left Atrial volume may be a more sensitive index of Left Atrial remodeling than Left Atrial dimension. Our study demonstrated an inverse correlation between maximum Left Atrial volume and systolic Strain as measured at atrial septum and Lateral Left Atrial wall in patients with severe Mitral Stenosis. Thus strain parameters may also be helpful in assessing Left Atrial remodeling and prediction of Atrial Fibrillation.

During ventricular systole, Atria functions as a reservoir to store blood when AV valve is closed. This reservoir function is postulated to be influenced by atrial relaxation, ventricular contraction through descent of the base and atrial stiffness[124]. A significant correlation between systolic atrial Strain and pulmonary vein peak systolic flow suggests that atrial peak systolic Strain is measure of atrial reservoir function [55,125,126]. Conversely only a small correlation has been found between peak systolic myocardial atrial Strain and ventricular peak systolic

Strain and no correlation at all has been found between the peak systolic AV ring displacement and atrial peak systolic deformation properties[56]. These findings confirm that the peak systolic atrial Strain and Strain Rate are less influenced by tethering effects and global heart motion and suggest that the abnormal atrial deformation properties during this phase are due mainly to changes in atrial myocardial compliance.

Atrial Deformation properties measured during diastole were not significantly different in patients with Mitral Stenosis when compared to normal individuals in our study. It may due to the fact that during diastole Mitral valve is wide open and left atrial function is also influenced by LV compliance. This has also been suggested by Di Salvo et al [56] who demonstrated a strong correlation between early diastolic Strain and Strain Rate values and LV global diastolic function indices and between peak early diastolic Strain and peak Systolic annular excursion. Also the lack of abnormalities in diastolic parameters may also be explained at least in part by their lower reproducibility as reported in earlier studies [56,116].

Taking together poor reproducibility and dependence of left atrial contractile function on factors other than atrial myocardial properties, diastolic Strain parameters may not be suitable for atrial dysfunction assessment. This may be the reason for lack of diastolic Strain parameters to identify contractile dysfunction in our study. Moreover several studies have demonstrated the

superiority of systolic atrial myocardial deformation properties as predictor of Atrial Fibrillation and cardiovascular events than diastolic atrial deformation parameters [56,57, 98,127] thus further limiting their application.

Present study also demonstrated lower diastolic Tissue Doppler velocities of atrial septum and Left Atrial lateral wall in severe Mitral Stenosis as compared to healthy individuals. Mi Seung Shin et al [115] in their study of 22 patients also reported lower peak systolic and late diastolic tissue velocities in patients with moderate to severe Mitral Stenosis as compared to controls, except that at septum. In our study even the velocities assessed at septum were lower in Mitral Stenosis group. This probably may be related to severity of Mitral Stenosis in patients included in the study. Mi Seung Shin et al [115] in their study included patients with moderate to severe stenosis, whereas in our study all patients has severe Mitral Stenosis which could have resulted in more severe left atrial dysfunction and thus abnormal septal Tissue Doppler velocities. Post Balloon Mitral Valvotomy early diastolic Tissue Doppler velocity of Lateral LA wall improved whereas that of Interatrial Septum remained unchanged in our study. This may be attributed to, interatrial septum being more as compared to lateral LA wall. Sudden decrease in left atrial after load after BMV improves Lateral wall velocity within 24 hours after BMV which is a relatively free structure.

SUMMARY: Our study demonstrates that atrial Tissue Doppler velocities & Tissue Doppler derived Strain imaging is a feasible method for assessment of left atrial function. It can be applied to evaluate impairment of atrial reservoir function in severe Mitral Stenosis. The degree of impairment is correlated to LA maximum volume which in turn is a sensitive predictor of cardiovascular events. Balloon Mitral Valvotomy tends to normalize these abnormalities within 24 hours after the procedure. Since these parameters are believed to predict cardiovascular events they may be useful to determine prognosis and identify patients at risk of complications as AF .This may also help to guide therapeutic decision in these patients.

LIMITATIONS

1) In our study we used Tissue Doppler derived Strain measurements which are known to be dependent on the Doppler angle of interrogation in relation to myocardial motion. Recently developed 2D speckle tracking imaging is independent of angle. However the thickness of the region of interest cannot be reduced to include extensively thin atrial walls in 2D speckle tracking imaging. Accordingly we selected Tissue Doppler imaging to measure tissue velocities and strain of thin atrial walls.

2) Left atrial wall particularly Interatrial septum is at times too thin, which may limit derivation of correct myocardial velocities and strain curves. We tried and kept left atrial walls well in view before applying Tissue Doppler to overcome this limitation.

3) Adequate visualization of Interatrial septum and lateral LA wall particularly in patients with severe Mitral Stenosis with enlarged left atrium was at times difficult and time consuming. Adequate visualization to obtain optimal tissue Doppler velocity and Strain curves often required aprox 30-40 minutes per patient thus making the modality prohibitively time consuming. This also decreases the reproducibility of the technique.

4) Clinical implications of abnormal strain parameters were not directly assessed in our study. However correlation between systolic Strain and LA maximum volume could be seen and LA volume is known to be a sensitive indicator of cardiovascular events.

5) Number of individuals included in the study was only 50 (25 Controls and 25 Mitral Stenosis). A larger study is needed to confirm our findings.

CONCLUSION

1) Left atrial function as assessed by Tissue Doppler velocities and Tissue Doppler derived Strain is abnormal in patients with severe Mitral Stenosis.

2) Abnormality of left atrial reservoir function as assessed by Tissue Doppler Strain imaging normalizes within 24 hours after Balloon Mitral Valvotomy.

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APPENDIX

ABBREVIATIONS

A4C: Apical Four Chamber View

BMV: Balloon Mitral Valvotomy

EF : Ejection Fraction

IAS : Interatrial Septum

LA : Left Atrium

LV : Left Ventricle

TR : Tricuspid Regurgitation

MS : Mitral Stenosis

MVA : Mitral Valve Area

WORKING PROFORMA

Name Hospital No. : Age / Sex : Date:

Diagnosis :

M mode Measurements (mm)

Aorta :…… RV :……
LA (PLAX) :…… IVS :…… PW:……
LVIDS :…… LVIDD:……

Doppler Data

Mitral E :……cm/sec Mitral A:……cm/sec
MR : AR: MVA: ……cm2
Aortic Gradient:……mmHg TR Gradient :……mmHg

2D Measurements

LVEDV:…… ml LVESV:……ml EF:…… % LA(A4C):……mm
LA vol (max):……..ml LA vol (min):……ml LAEF: ……..%
MVA:.....cm2

Tissue Doppler Measurements

	Lateral Wall	IAS
E' Velocity (cm/sec)		
A' Velocity (cm/sec)		
Strain at ventricular end systole (%)		
Strain at ventricular late Diastole (%)		
Strain rate at ventricular end systole (per sec)		
Strain rate at ventricular late Diastole (per sec)		

GLOSSARY FOR MASTER CHART

Aorta	- M Mode Aorta Dimensions in mm
LA	- M Mode Left Atrial Dimensions in mm
RV	- M Mode Right Ventricular Dimensions in mm
IVS	- M Mode Inter Ventricular Septum Dimensions in mm
PW	- M Mode Posterior wall Dimensions in mm
LVIDD	- M Mode LV Dimensions in diastole in mm
LVIDS	-M Mode LV Dimensions in systole in mm
E	-Doppler Mitral E Velocity in cm/sec
A	-Doppler Mitral A Velocity in cm/sec
MR	-Doppler Mitral Regurgitation
AR	-Doppler Aortic Regurgitation
AoGd	-Doppler Peak Aortic Gradient in mmHg
TRGd	-Doppler Peak TR gradient in mmHg
MVAdop	-Mitral Valve Area by Doppler in cm^2
LVEDV	-2D LV end diastolic volume in ml
LVESV	-2D LV end systolic volume in ml
LVEF	-LV EF by Simpson's method
LAmax	-2 D Left Atrial maximum volume in ml
LAmin	-2 D Left Atrial minimum volume in ml
LAEF	-Left Atrial EF by Simpson's Method
LAsize	-Maximum LA size in A4C in mm
MVA2D	-Mitral Valve Area by 2D in cm^2

IASE -IAS pulse wave E' Velocity

IASA -IAS pulse wave A' Velocity

IASSES -IAS Strain at ventricular end systole (%)

IASSLD -IAS Strain at ventricular late diastole (%)

IASSRES -IAS Strain Rate at ventricular end systole (per sec)

IASSRLD -IAS Strain Rate at ventricular Late Diastole (per sec)

LATE - Lateral Atrial Wall pulse wave E' Velocity

LATA -Lateral Atrial Wall pulse wave A' Velocity

LATSES -Left Atrial Lateral Wall strain at ventricular end systole(%)

LATSLD -Left Atrial Lateral Wall strain at ventricular Late Diastole(%)

LATSRES -Left Atrial Lateral Wall strain rate at ventricular end systole (per sec)

LATSRLD -Left Atrial Lateral Wall strain rate at ventricular Late Diastole (per sec)

pAorta -Post BMV M Mode Aorta Dimensions in mm

pLA -Post BMV M Mode Left Atrial Dimensions in mm

pRV -Post BMV M Mode Right Ventricular Dimensions in mm

pIVS -Post BMV M Mode Inter Ventricular Septum Dimensions in mm

pPW -Post BMV M Mode Posterior wall Dimensions in mm

pLVIDD -Post BMV M Mode LV Dimensions in diastole in mm

pLVIDS -Post BMV M Mode LV Dimensions in systole in mm

pMR -Post BMV Doppler Mitral Regurgitation

pAR -Post BMV Doppler Aortic Regurgitation

pAoGd -Post BMV Doppler Peak Aortic Gradient in mmHg

pTRGd -Post BMV Doppler Peak TR gradient in mmHg

pMVADop -Post BMV Mitral Valve Area by Doppler in cm2

pLVEDV -Post BMV 2D LV end diastolic volume in ml

pLVESV -Post BMV 2D LV end systolic volume in ml

pLVEF -Post BMV LV EF by Simpson's method

pLAmax -Post BMV 2 D Left Atrial maximum volume in ml

pLAmin -Post BMV 2 D Left Atrial minimum volume in ml

pLAEF -Post BMV Left Atrial EF by Simpson's Method

pLAsize -Post BMV Maximum LA size in A4C in mm

pMVA2D -Post BMV Mitral Valve Area by 2D in cm^2

pIASE -Post BMV IAS pulse wave E' Velocity

pIASA -Post BMV IAS pulse wave A' Velocity

pIASSES -Post BMV IAS Strain at ventricular end systole (%)

pIASSLD -Post BMV IAS Strain at ventricular late diastole (%)

pIASSRES -Post BMV IAS Strain Rate at ventricular end systole (per sec)

pIASSRLD -Post BMV IAS Strain Rate at ventricular Late Diastole (per sec)

pLATE -Post BMV Lateral Atrial Wall pulse wave E' Velocity

pLATA -Post BMV Lateral Atrial Wall pulse wave A' Velocity

pLATSES -Post BMV Left Atrial Lateral Wall strain at ventricular end systole(%)

pLATSLD -Post BMV Left Atrial Lateral Wall strain at ventricular Late Diastole(%)

pLATSRES -Post BMV Left Atrial Lateral Wall strain rate at ventricular end systole (per sec)

pLATSRLD -Post BMV Left Atrial Lateral Wall strain rate at ventricular Late Diastole (per sec)

S.NO.	NAME	AGE	SEX	HOSP.NO.	Group	Aorta	LA	RV	IVS	PW	LVIDD	LVIDS	E	A	MR	AR	Ao Gd	TRGd	MVAdop	LVEDV
1	Jonaed Iqbal	20	Male	379863D	Control	17	21	12	8.3	8.3	44.2	28	80	40	Nil	Nil	5	20	.	40
2	Bidyut Saman	27	Male	402744D	Control	23	35	23.8	12.1	12.1	40.5	27.6	90.3	59.2	Nil	Nil	7	20	.	114
3	Chandan Sadh	32	Male	407419D	Control	22	32	14.6	11	10.6	48	29.5	88.1	64.7	Nil	Nil	5	17	.	101
4	Pazhani	39	Male	407470D	Control	20	33	12.1	9.1	8.7	43.5	26.8	81.9	71.7	Nil	Nil	10	25	.	110
5	Dayan Nab	39	Male	409873D	Control	22	27	12.9	8.7	8.7	46.1	27.6	75.5	60.2	Nil	Nil	5	17	.	79
6	Saravanan	34	Male	328893D	Control	21	24	9.4	8.3	9	43.5	30.2	94.3	67.2	Nil	Nil	7	.	.	75
7	Satyajit Das	12	Male	405892D	Control	17	22	15.9	8.3	7.9	38.6	25	103	64.7	Nil	Nil	7	20	.	69
8	Alok Ghosh	24	Male	412649D	Control	27	28	26.5	9.8	9	42	29.5	101	54.8	Nil	Nil	8	.	.	100
9	Dilip Kumar	33	Male	456289D	Control	23	34	12.5	10.6	11.7	44.2	28.7	86.9	73.1	Nil	Nil	6	.	.	79
10	Rajeswar Shaw	34	Male	988513B	Control	23	35	20	9.8	9.8	43.9	29.1	63.9	51.3	Nil	Nil	4	16	.	111
11	Yesaianl	49	Male	390896D	Control	28	29	9	8.3	8.7	43.1	28	75	70.1	Nil	Nil	6	.	.	78
12	Samiran Dutta	30	Male	458580D	Control	20	25	19.3	9.2	8.9	39.3	26.1	79.5	59.7	Trace	Nil	7	9	.	61
13	Sumathy	30	Female	423363D	Control	19	32	8.3	7.9	7.9	44.2	30.6	89.3	84.4	Nil	Nil	11	14	.	77
14	Sudeshna Ghoshal	31	Female	464054D	Control	20	30	17	9.4	8.7	40.8	26.8	99.2	59.2	Nil	Nil	8	4	.	97
15	Asesh Kumar	49	Male	463296D	Control	22	32	15.1	10.2	9.8	37.1	23.8	85.4	85.4	Nil	Nil	7	.	.	79
16	Anil Kumar	43	Male	464666D	Control	26	30	14.4	10.6	11	34.8	23.8	61.2	56.3	Trace	Nil	4	18	.	77
17	Jakhir Khan	35	Male	424638D	Control	24	37	15.9	11	11	36.8	25.7	90.3	58.2	Trace	Nil	5	14	.	101
18	Rajeswari	29	Female	491845D	Control	19	23	13.2	7.5	7.1	34.4	23.8	78	54.8	Nil	Nil	5	17	.	61
19	Shanthi	35	Female	334282D	Control	23	26	19.7	8.2	7.5	43.2	29	80.5	70.1	Nil	Nil	7	.	.	72
20	Abu Sauban	25	Male	547951D	Control	21	32	15.5	8.7	8.3	42	27	93.8	71.6	Nil	Nil	6	16	.	71
21	Partha Pratim	28	Male	547144D	Control	23	28	14.7	10	9.6	47	35	71.1	54.3	Nil	Nil	5	17	.	68
22	Praful Kumar	24	Male	528357D	Control	24	31	12.7	8.5	9.4	44	30	81.9	53.3	Nil	Nil	3	24	.	108
23	Manoj Kumar	17	Male	458118D	Control	22	29	13.2	9.4	8.7	45	30	103	83	Nil	Nil	5	20	.	79
24	Amitava	23	Male	527906D	Control	22	26	12.5	8.7	8.3	38	28	80.5	70.1	Nil	Nil	5	21	.	75
25	Keshab	26	Male	406950B	Control	24	27	17.8	9	9	43.1	29.5	91.9	61.3	Nil	Nil	7	.	.	90
26	Snowmala Deb	38	Female	387511D	Mitral Stenosis	20	50	20	10.2	10.6	45.4	31.8	.	.	Nil	Trace	7	32	0.98	74
27	Manju Devi	27	Female	390237D	Mitral Stenosis	18	43	16.6	9.8	10.6	37.1	25	.	.	Nil	Nil	4	49	0.51	55
28	Mira Mondal	30	Female	391570D	Mitral Stenosis	18	37	12	9.8	9.4	41.2	26.5	.	.	Nil	Nil	10	35	0.94	45
29	Briendra	39	Male	390241D	Mitral Stenosis	22	55	25	11.7	10.6	34	22.7	.	.	Nil	Nil	6	125	0.72	34
30	Anjala	30	Female	399590D	Mitral Stenosis	16	30	22.3	8.3	8.3	32.1	22.3	.	.	Trace	Nil	8	42	1.04	54
31	Venkatesan	38	Male	394172D	Mitral Stenosis	20	35	15.9	10.3	9.6	32.4	20.8	.	.	Nil	Nil	5	72	0.88	22
32	Rosalin	28	Female	380912D	Mitral Stenosis	20	48	14.7	11.3	10	34	24.2	.	.	Nil	Nil	11	80	0.79	47
33	Lalitha	31	Female	398350D	Mitral Stenosis	19	47	12.5	8.3	7.5	36.7	23.4	.	.	Nil	Nil	7	51	0.88	50
34	ReenaPatra	30	Female	396484D	Mitral Stenosis	19	42	11	8.3	7.9	41.6	27.2	.	.	Mild	Nil	8	31	0.86	61
35	Tapeswar Prasad	36	Male	399101D	Mitral Stenosis	21	48	10.2	9	9.8	47.3	32.5	.	.	Nil	Nil	5	69	0.91	30
36	Md Tarwez	25	Male	402652D	Mitral Stenosis	20	47	12.3	9.8	9.8	49.8	31.5	.	.	Nil	Trace	8	61	0.89	87
37	Tamil Selvi	27	Female	531759C	Mitral Stenosis	18	44	17.8	9.8	9.1	48.8	32.9	.	.	Nil	Nil	8	38	1.04	93
38	Geetha Kumari	24	Female	447980D	Mitral Stenosis	22	50	15.9	8.7	8.3	41.6	29.5	.	.	Trace	Mild	9	76	0.61	52
39	Murugan V	24	Male	443193D	Mitral Stenosis	21	41	14.7	9.4	9.8	37.1	26.1	.	.	Nil	Trace	9	104	0.65	53
40	Poonkodi	22	Female	325827D	Mitral Stenosis	19	25	10.6	8.2	7.9	27.1	19.2	.	.	Nil	Trace	4	13	1.07	47
41	Uttam Kumar	17	Male	749569C	Mitral Stenosis	22	42	11	9.8	9.4	41.6	27.6	.	.	Nil	Nil	7	20	0.58	79
42	Minnu S	19	Female	460224D	Mitral Stenosis	19	36	13.6	8.7	8.7	35.2	23.4	.	.	Nil	Nil	7	82	0.91	60
43	Suresh Jaiswal	27	Male	422477C	Mitral Stenosis	19	47	11.3	9.8	9.8	43.1	29.9	.	.	Nil	Trace	14	26	0.94	67
44	Bhuneswari Devi	25	Female	462956D	Mitral Stenosis	21	33	14.6	8.6	8.6	45.7	28.5	.	.	Trace	Nil	8	35	0.73	54
45	Birendra Yadao	39	Male	390241D	Mitral Stenosis	22	55	25	11.7	10.6	34	22.7	.	.	Nil	Nil	6	125	0.72	34
46	Lakshmi	41	Female	352055D	Mitral Stenosis	21	33	16.3	7.1	7.1	43.5	29.1	.	.	Trace	Nil	6	34	0.95	77
47	Devayanai Patra	23	Female	522561D	Mitral Stenosis	19	44	12.5	8.3	8.3	37.4	25.9	.	.	Trace	Nil	10	5	0.76	60
48	Lalita Mondal	19	Female	569264D	Mitral Stenosis	20	48	10.2	9.4	9	40.1	27.6	.	.	Nil	Nil	4	79	0.79	56
49	Chandani Pati	33	Female	533476C	Mitral Stenosis	19	42	11.8	8.6	8.6	40.8	27	.	.	Trace	Nil	8	26	0.91	79
50	Shipra Das	26	Female	602133D	Mitral Stenosis	22	38	11	9.8	9.4	47.3	32.5	.	.	Nil	0	12	65	0.85	109

S.NO.	NAME	LVESV	LVEF	Lamax	Lamin	LAEF	LA size	MVA2D	IASE	IASA	IASSES	IASSLD	IASSRES	IASSRLD	LATE	LATA	LATSES	LATSLD	LATRES
1	Jonaed Iqbal	13	68	46	20	57	43	.	9.75	6.04	28.25	0.74	-0.65	-0.37	16.8	6.92	30.39	-0.5	0.51
2	Bidyut Saman	46	60	72	28	61	55	.	8.24	13.8	25.95	-1	-4	-0.02	10.1	9.02	5.35	-0.61	0.99
3	Chandan Sadh	42	58	36	11	69	43.5	.	10.4	7.3	36.54	0.3	1.01	0.29	16	18.5	33.31	-0.21	0.78
4	Pazhani	38	65	46	16	65	53.5	.	8.5	13.5	27.99	-0.29	-0.47	-0.03	11.3	15.5	15	0.12	-0.57
5	Dayan Nab	31	61	28	12	54	43.5	.	10.6	13.8	32.38	-0.19	0.6	-0.12	11.7	15.2	33.84	-0.18	0.79
6	Saravanan	25	67	42	20	52	43.4	.	13.7	5.6	30.14	0.24	-1.6	0.17	19	10.1	33.37	-0.01	1.03
7	Satyajit Das	24	65	30	10	67	46.4	.	9.69	7.91	55.65	-0.3	-0.36	0.2	17.4	13.8	57.39	2.88	-1.5
8	Alok Ghosh	42	58	37	16	57	45.9	.	9.55	8.86	40.03	-0.83	0.63	0.09	18.4	17.1	8.99	0.01	0.1
9	Dilip Kumar	32	59	40	20	50	48.3	.	7.99	11.6	40.3	0	1.38	-0.41	14.5	16.5	28.67	0	2.07
10	Rajeswar Shaw	46	59	43	22	49	49.8	.	7.6	10.3	23.76	0	-0.24	0.07	13.3	7.6	32.47	0	0.14
11	Yesaianl	31	60	22	11	50	40.3	.	6.14	9.65	22.26	0	-0.29	0.21	12.1	14.9	31.56	0	-0.18
12	Samiran Dutta	25	59	28	12	57	41.7	.	12.4	8.58	21.12	0	-0.05	0.77	15.1	9.75	7.51	0	0.44
13	Sumathy	32	58	27	14	48	40	.	7.31	9.26	21.34	0	0.51	0.49	13.6	12.2	46.54	0	0.11
14	Sudeshna Ghoshal	36	63	42	18	57	44.7	.	7.6	6.34	20.59	0	-0.09	0.19	16.2	13.7	11.11	0	0.54
15	Asesh Kumar	34	57	35	17	54	45.7	.	13.9	16.6	30.58	0	-0.12	0.11	15.1	19.7	31.8	0	0.12
16	Anil Kumar	32	56	26	10	62	42.8	.	7.6	9.16	24.09	-0.74	-0.17	0.16	10.1	12.3	24.55	0.37	-0.04
17	Jakhir Khan	38	62	43	18	58	55.7	.	14.7	10.1	34.64	0	-0.32	0.91	20.1	16.2	9.3	0	-0.1
18	Rajeswari	23	62	25	9	64	38.9	.	10.2	5.56	58.87	0	-0.04	0.36	20.3	11.7	33.01	0	-0.04
19	Shanthi	26	64	25	12	52	46	.	12	7.31	23.86	0	0.54	0.15	17.9	16	20.01	0	-0.29
20	Abu Sauban	27	62	33	14	58	41.8	.	8.19	5.07	22.02	0	-0.15	0.49	18.2	11.4	26.61	0	0.98
21	Partha Pratim	24	65	26	10	62	48.7	.	6.24	7.9	16.61	0	0.32	0.27	14.3	10.1	42.75	0	0.44
22	Praful Kumar	43	60	38	13	66	47	.	8.58	7.21	19.84	0.96	-0.47	1.14	13	9.65	45.28	0	-0.1
23	Manoj Kumar	32	59	35	12	66	41.4	.	7.5	11.7	30.76	0	-0.44	-1.4	21.3	16.6	39.97	0	1.26
24	Amitava	21	61	23	7	70	41	.	9.36	6.63	32.19	0	0.64	1.47	13.9	7.7	46.78	0	1.01
25	Keshab	38	58	36	17	53	51.4	.	16.2	10.2	20.12	0	-0.33	0.66	29.9	14.5	15.56	0	-0.04
26	Snowmala Deb	25	66	167	133	20	83.5	0.93	3.41	6.82	11.53	0.74	1.57	-0.01	10.5	7.31	18.6	0.24	-0.11
27	Manju Devi	21	62	58	44	24	65	0.9	3.19	5.33	7.19	0.66	-0.16	0.04	3.33	3.43	17.91	-0.07	0.7
28	Mira Mondal	20	56	70	42	40	57.7	1.07	5.3	7.51	0.85	0.67	-0.87	-0.19	5.1	7.8	16.89	-0.24	0.35
29	Briendra	11	68	113	94	18	65.6	0.84	8.8	6.46	15.37	-0.01	-0.75	-0.26	5.46	7.24	8.2	0.61	0.44
30	Anjala	18	67	50	35	30	54.7	0.99	12.7	7.8	23.34	0.13	-1.5	-0.22	11	7.8	21.62	0.11	0.09
31	Venkatesan	8	64	86	57	29	63.2	1.24	8.91	6.35	2	-0.29	0.86	-0.34	7.02	6.02	8.88	-0.14	0.84
32	Rosalin	20	57	96	85	11	67.5	0.64	10	6.68	7.28	0.03	0.82	-0.01	5.6	8.13	12.9	0	-0.42
33	Lalitha	18	64	85	70	13	66.9	0.9	9.69	7.69	15.89	-0.11	1.33	-0.08	8.02	7.02	23.47	0.35	-0.8
34	ReenaPatra	20	67	75	52	29	55.9	0.82	17.2	13.5	5.21	-0.43	-0.59	-0.28	7.5	13.4	26.41	-0.33	1.37
35	Tapeswar Prasad	11	63	110	90	18	59.8	0.96	8.7	10.3	7.69	-0.33	0.52	-0.13	6.14	5.56	16.75	0.06	-0.25
36	Md Tarwez	33	62	113	86	24	72.9	0.78	8.29	11.7	5.49	0.33	0.76	-0.37	7.9	4.58	21.86	0	0.49
37	Tamil Selvi	36	61	87	66	24	60.8	0.8	4.09	6.53	6.22	0	0.1	-1.3	4.78	7.41	32.77	0	0.97
38	Geetha Kumari	22	58	115	95	17	70.6	0.74	5.7	5.07	10.55	0	-0.36	0.18	6.4	3.4	-6.25	-0.6	0.44
39	Murugan V	18	66	97	83	14	70.2	0.9	4.19	5.85	17.07	0	1.44	-0.03	6.53	7.6	6.23	0	-0.46
40	Poonkodi	16	66	29	18	28	44.4	0.91	6.14	8.38	7.83	-0.66	0.15	0.36	10.6	7.9	33.1	-1.4	-0.18
41	Uttam Kumar	29	63	70	52	26	65.2	0.77	5.6	3.43	13.11	0	0.03	0.31	7.27	4.85	27.02	0	-0.97
42	Minnu S	26	57	87	65	25	69.9	0.8	3.62	2.97	6.21	0.22	0.17	-0.06	5.46	4	25.33	2.08	0.14
43	Suresh Jaiswal	28	58	70	46	34	51.6	0.79	10.2	9.94	15.8	0	-1.9	-0.03	6.24	8.29	20.82	0	0.44
44	Bhuneswari Devi	20	63	39	30	23	58.4	0.67	2.42	5.22	16.94	-0.09	0.32	-0.29	4.76	6.86	10.64	-0.47	1.26
45	Birendra Yadao	11	68	113	94	18	63	0.84	8.8	6.4	8.61	0.43	0.99	0.23	5.4	7.2	3.75	0.14	-0.63
46	Lakshmi	33	57	145	114	21	65.7	1.07	6.35	8.24	14.95	0	-0.04	-0.57	6.13	8.58	15.49	0	0.09
47	Devayanai Patra	24	60	112	85	24	76.7	0.9	3.51	4.78	17.61	0.01	-0.14	-0.11	4.97	8.97	16.08	0	0.05
48	Lalita Mondal	21	63	113	88	22	66.8	0.84	2.53	3.61	7.05	0	0.18	-0.77	4.29	5.95	6.05	0	-0.04
49	Chandani Pati	31	67	86	67	22	67.3	0.99	8.48	10.7	15.27	0	0.32	0.27	8.36	16.9	31.7	0	-0.42
50	Shipra Das	46	58	79	55	30	66	0.67	4.39	6.04	26.26	0	0.49	-0.33	6.21	8.97	26.97	0	0.28

S.NO.	NAME	LATSRLD	pAORTA	pLA	pRV	pIVS	pPW	pLVIDD	pLVIDS	pMR	pAR	pAoGd	pTRGd	pMVADop	pLVEDV	pLVESV	pLVEF	pLAmax	pLAmin
1	Jonaed Iqbal	0.28	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
2	Bidyut Saman	0.15	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
3	Chandan Sadh	0.32	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
4	Pazhani	-0.09	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
5	Dayan Nab	0.07	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
6	Saravanan	0.01	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
7	Satyajit Das	-12	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
8	Alok Ghosh	0.04	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
9	Dilip Kumar	-0.63	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
10	Rajeswar Shaw	0.21	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
11	Yesaianl	0.16	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
12	Samiran Dutta	0.04	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
13	Sumathy	-0.13	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
14	Sudeshna Ghoshal	-0.63	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
15	Asesh Kumar	0.76	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
16	Anil Kumar	-0.21	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
17	Jakhir Khan	-0.08	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
18	Rajeswari	0.28	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
19	Shanthi	0.31	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
20	Abu Sauban	0.13	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
21	Partha Pratim	0.72	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
22	Praful Kumar	0.46	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
23	Manoj Kumar	0.13	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
24	Amitava	1.26	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
25	Keshab	-0.14	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
26	Snowmala Deb	-0.05	19	47	10.6	11	10.6	44.2	26.8	Nil	Trace	12	18	1.7	73	26	64	136	89
27	Manju Devi	-0.1	20	37	8.3	8.7	9	41.2	25.7	Mild	Nil	4	50	2.2	58	21	63	52	42
28	Mira Mondal	-0.05	22	34	17.8	10	9.8	40.5	25.7	Nil	Nil	10	28	2.2	62	26	58	50	31
29	Briendra	-0.25	22	42	30	10	10	39	26	Nil	Nil	6	41	1.65	46	15	57	68	47
30	Anjala	-0.12	16	31	15.9	8.3	8.3	34	22.3	Nil	Nil	10	39	2.04	47	17	64	42	20
31	Venkatesan	0.13	25	34	19.3	11	9.4	30.6	21.2	Nil	Nil	8	69	1.69	37	13	65	66	56
32	Rosalin	-0.04	19	46	14.4	10.6	9.4	34.4	23.8	Nil	Nil	10	40	1.9	56	23	59	59	47
33	Lalitha	0.43	19	42	12.1	7.9	7.1	36.7	23.8	Nil	Nil	5	21	1.88	73	28	62	74	61
34	ReenaPatra	0.08	18	42	11	7.5	7.5	40.5	26.8	Mild	Nil	8	27	2.24	76	28	68	70	38
35	Tapeswar Prasad	0.34	21	44	9.07	9.07	9.8	48.4	31.8	Nil	Nil	8	32	1.58	69	26	62	80	54
36	Md Tarwez	0.07	21	43	14	11.3	11.3	48	32.1	Nil	Nil	6	40	2.02	82	32	61	117	74
37	Tamil Selvi	0.68	21	36	9.83	7.56	8.32	50.3	34.8	Nil	Nil	8	22	1.68	107	45	58	59	42
38	Geetha Kumari	-0.76	19	46	10.2	8.32	8.32	46.5	31.8	Nil	Mild	10	54	1.68	74	32	57	112	93
39	Murugan V	-0.23	22	30	12.9	8.8	8.2	42.5	28.6	Trace	Trace	10	22	1.91	86	33	62	64	48
40	Poonkodi	-0.1	18	22	9.9	7.8	8.4	31.8	22.1	Trace	Trace	3	8	1.95	56	22	60	27	16
41	Uttam Kumar	0.58	21	40	9	8.3	8.3	44.2	28	Trace	Nil	8	33	1.86	77	29	62	57	38
42	Minnu S	-1.7	17	33	15.2	10.6	9.9	38.7	26.5	Mild	Trace	5	59	2.44	53	19	64	46	30
43	Suresh Jaiswal	-0.4	18	36	12.1	8.3	8.3	40.1	28	Nil	Nil	18	22	1.93	86	35	61	69	44
44	Bhuneswari Devi	-0.56	20	30	15.7	8.2	8.5	40.4	27.9	Nil	Nil	10	14	2.29	57	24	58	32	22
45	Birendra Yadao	-0.24	22	42	22	11	10	33	23	Nil	Nil	6	41	1.65	46	15	67	68	47
46	Lakshmi	-0.12	22	50	11	8.6	8.2	42.5	29.4	Trace	Nil	9	29	1.8	94	36	62	99	61
47	Devayanai Patra	-0.72	17	41	10.2	7.5	7.5	38.9	25.7	Mild	Nil	11	5	1.68	57	20	65	95	66
48	Lalita Mondal	0.04	23	36	8.3	8.7	7.9	44.2	29.1	Trace	Nil	5	27	1.83	53	21	60	97	66
49	Chandani Pati	-0.37	24	35	7.3	7.7	7	40.7	27.7	Nil	Nil	8	22	1.64	101	39	61	85	47
50	Shipra Das	0.5	19	31	11.4	10	9.6	45	31.8	Trace	Nil	10	29	1.88	90	37	59	50	25

S.NO.	NAME	pLAEF	pLAsize	pMVA2D	pIASE	pIASA	pIASSES	pIASSLD	pIASSRES	pIASSRLD	pLATE	pLATA	pLATSES	pLATSLD	pLATSRES	pLATSRLD
1	Jonaed Iqbal	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
2	Bidyut Saman	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
3	Chandan Sadh	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
4	Pazhani	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
5	Dayan Nab	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
6	Saravanan	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
7	Satyajit Das	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
8	Alok Ghosh	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
9	Dilip Kumar	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
10	Rajeswar Shaw	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
11	Yesaianl	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
12	Samiran Dutta	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
13	Sumathy	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
14	Sudeshna Ghoshal	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
15	Asesh Kumar	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
16	Anil Kumar	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
17	Jakhir Khan	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
18	Rajeswari	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
19	Shanthi	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
20	Abu Sauban	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
21	Partha Pratim	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
22	Praful Kumar	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
23	Manoj Kumar	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
24	Amitava	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
25	Keshab	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
26	Snowmala Deb	35	74.3	2.1	8.47	7.46	22.33	0.8	0.17	0.22	11	8.58	23.83	1.84	-0.27	-0.68
27	Manju Devi	21	63.2	1.97	4	4.19	17.97	1.92	-0.09	0.06	9.9	9.85	16.53	-1.1	-0.17	0.14
28	Mira Mondal	38	53	1.82	8.58	4.19	15.12	-0.87	-0.1	-0.11	6.92	6.53	36.12	-0.26	-0.32	0.18
29	Briendra	31	59.7	1.75	4.01	8.36	15.49	0.35	0.13	-0.49	10.6	9.02	17.79	-0.19	-0.75	-0.58
30	Anjala	52	60.7	1.86	5.26	8.58	2.42	-0.09	0.64	-0.07	9.07	10.1	8.62	0.17	-0.36	0.13
31	Venkatesan	14	63.9	1.86	4.68	6.82	9.33	0.02	-0.43	0.15	5.36	6.73	6.18	0.1	-0.01	-0.04
32	Rosalin	20	63.2	1.73	6.04	5.85	30.47	1.48	0.61	-0.63	8.97	13.8	39.21	-0.43	-1.1	-1.27
33	Lalitha	18	65	2.05	5.35	4.79	35.5	0.5	-0.47	0.03	8.47	5.35	27.87	2.15	-1.6	0.56
34	ReenaPatra	46	50.5	2.86	15.1	10.7	49.57	-0.71	1.71	-0.09	12.7	7.4	37.35	-0.31	-0.37	0.01
35	Tapeswar Prasad	33	57.3	2.14	6.8	8.36	20.28	-0.73	0.59	-0.11	5.24	6.02	11.38	0.03	-1.2	0.02
36	Md Tarwez	37	75	1.74	9.25	7.58	10.32	0.33	0.54	0.33	10	4.79	13.63	0.21	0.18	-0.04
37	Tamil Selvi	29	60.4	1.9	5.65	7.12	4.14	-0.99	0.4	-1.2	9.55	8.19	21.43	0	0.37	-0.41
38	Geetha Kumari	17	64.7	1.9	3.41	6.24	7.17	1.66	0.28	-1.3	5.65	4.87	15.76	-0.24	0.56	0.13
39	Murugan V	25	69.2	2	3.31	5.95	14.5	0	0.05	0.46	5.56	5.36	32.9	0	0.29	0.43
40	Poonkodi	43	48.8	1.97	5.56	5.07	20.19	0	-0.17	1.31	11.5	9.36	17.41	-4.2	-0.73	0.12
41	Uttam Kumar	33	58.4	2.1	7.21	3.22	33.9	0	0.98	0.42	9.85	5.46	19.57	0	0.41	1.26
42	Minnu S	37	65.4	1.56	6.14	2.92	11.13	-1.7	-0.17	1.18	8.58	5.17	16.39	-0.53	-0.06	-0.5
43	Suresh Jaiswal	36	61	1.95	5.85	7.14	16.02	0	0.14	-0.23	7.8	8.87	33.27	0	0.79	0.66
44	Bhuneswari Devi	31	57.1	2.03	7.99	4.58	7.6	0	-0.48	-1.4	9.55	5.17	13.58	0	-0.4	0.28
45	Birendra Yadao	31	59	1.75	4.01	8.36	14.06	1.18	0.38	-0.17	10.6	9.02	4.4	-0.28	-1.2	-0.16
46	Lakshmi	38	76	2	7.7	6.14	23.22	0	-0.09	-0.67	5.56	10.9	32.83	0	-0.19	-0.6
47	Devayanai Patra	31	73.8	1.52	6.14	7.21	12.73	0	0.38	-0.97	8.97	6.63	31.39	0	-0.37	0.53
48	Lalita Mondal	32	64.1	1.89	10.7	7.9	23.15	0	-0.1	-0.31	9.75	6.24	28.87	0	0.27	0.52
49	Chandani Pati	45	54	1.9	11.3	13.9	17.34	0	0.7	0.19	8.32	10.9	26.14	0	0.33	0.54
50	Shipra Das	50	58.8	2.11	5.07	7.02	15.33	0	0.19	-0.92	10.2	4.29	39.7	0	0.7	-1.17